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Imaging-Based Changes in Visceral Adipose Tissue and Metabolic Risk: A Systematic Review

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Background:

Visceral adipose tissue is a high-risk fat compartment linked to insulin resistance, dyslipidaemia, and hepatic steatosis. Imaging enables direct quantification of visceral adipose tissue and depot-specific change.

Methods:

PubMed was searched for human clinical trials and longitudinal cohorts reporting serial visceral adipose tissue quantified by magnetic resonance imaging, ultrasonography, or dual-energy X-ray absorptiometry and at least one metabolic outcome. Reference lists were hand-searched, screening and data extraction were performed in duplicate, and findings were synthesised narratively without meta-analysis.

Results:

Thirteen studies (10 trials, 3 cohorts; sample size 32–598; follow-up 8 weeks–2 years) were included, most commonly reporting glycaemic or insulin-resistance indices, lipid profile measures, and hepatic fat. In a cohort, each 10 cm^2 increase in visceral adipose tissue was associated with higher odds of metabolic syndrome (odds ratio 1.23; 95% confidence interval 1.09–1.39). In randomised trials, dapagliflozin reduced visceral adipose tissue volume by 0.35 L and liver fat by 3.74 percentage points versus placebo (8 weeks), and semaglutide reduced visceral fat mass by 27.4% versus 2.4% (68 weeks).

Conclusions:

Imaging-detected reductions in visceral adipose tissue were generally accompanied by improved metabolic risk markers, with the most consistent co-improvements in hepatic fat and lipid risk. Standardised imaging protocols and longer multicentre studies are needed to define clinically meaningful thresholds of visceral adipose tissue change across modalities.

Keywords:

Visceral Fat, Magnetic Resonance Imaging, Ultrasonography, Absorptiometry, Photon, Insulin Resistance, Metabolic Syndrome.

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Introduction

Excess visceral adipose tissue (VAT) is increasingly recognised as a high-risk adiposity phenotype that helps explain why individuals with similar body mass index (BMI) can have markedly different cardiometabolic trajectories. Compared with predominantly subcutaneous fat, VAT is more strongly linked to adverse adipokine profiles, low-grade inflammation, insulin resistance, and residual cardiovascular risk, making it a clinically meaningful target for prevention and treatment strategies that aim to reduce metabolic complications rather than weight alone [1]. In parallel, the field has shifted from surrogate anthropometry (for example, waist circumference) toward imaging-derived quantification of VAT, because imaging can distinguish visceral from subcutaneous compartments and provide a more direct assessment of biologically relevant fat distribution [2].

Longitudinal and interventional evidence supports VAT as a predictor and modifiable determinant of metabolic risk. In a large cohort followed for a median of 4.8 years, higher computed-tomography-derived visceral fat area (VFA) and visceral-to-subcutaneous fat ratio were independently associated with incident type 2 diabetes; compared with the lowest quartile, the highest VFA quartile showed adjusted odds ratios of 2.62 (95% confidence interval 1.73-3.97) in men and 32.49 (95% confidence interval 7.42-142.02) in women, with sex-specific reference thresholds proposed (VFA ≥130 cm^2 in men; ≥85 cm^2 in women) [3]. Beyond baseline prediction, pooled magnetic resonance imaging (MRI) analyses across dietary randomised controlled trials suggest that changes in visceral depots (expressed as VAT area and proportional distribution) relate to concurrent shifts in cardiometabolic profiles, reinforcing the rationale for focusing on imaging-based VAT change as an outcome in mechanistic and therapeutic studies [4]. The public-health importance of VAT-targeted strategies is underscored by the global expansion of overweight and obesity. A comprehensive pooled analysis of 3,663 population-representative studies covering 222 million participants reported that combined underweight and obesity prevalence increased in 162 of 200 countries (81%) among women and 140 of 200 countries (70%) among men from 1990 to 2022, illustrating a widespread rise in unhealthy weight extremes across regions and age groups [5]. This epidemiologic transition has been accompanied by substantial downstream morbidity, with an increasing proportion of cardiometabolic disease attributable to excess adiposity and adverse fat distribution, highlighting the need for scalable risk stratification methods that move beyond BMI and capture high-risk phenotypes such as visceral adiposity [1,5].

Consistent with these trends, Global Burden of Disease analyses show a growing worldwide burden attributable to high BMI, with global deaths and disability-adjusted life years more than 2.5-fold higher in 2021 than in 1990 for both sexes; age-standardised death rates were stable in females but increased by 15.0% in males, while age-standardised disability-adjusted life-year rates increased by 21.7% in females and 31.2% in males [6]. In 2021, the leading causes of high-BMI-attributable disability-adjusted life years included diabetes mellitus, ischaemic heart disease, hypertensive heart disease, chronic kidney disease, low back pain, and stroke, reinforcing that adiposity-related risk is mediated through multi-system pathways that are plausibly amplified by visceral fat biology [6]. Given VAT's established links to glycaemic dysregulation, atherogenic dyslipidaemia, blood pressure elevation, and inflammatory activation, imaging-quantified VAT offers a clinically relevant intermediate phenotype for evaluating both risk and the response to intervention,

particularly where hard outcomes require long follow-up [1,3,6]. A critical methodological challenge is that VAT can be assessed by multiple imaging modalities with differing acquisition protocols, anatomical definitions, and analytic pipelines, complicating synthesis across studies. MRI is frequently treated as a reference-standard method for compartmental adipose quantification in clinical research due to its high soft-tissue contrast and lack of ionising radiation, but its costs and availability constrain routine use [2]. Ultrasonography is attractive for its accessibility and feasibility at point of care; contemporary guidance highlights substantial heterogeneity in ultrasound measurement sites and constructs (for example, preperitoneal thickness versus intra-abdominal distances), which can affect comparability and clinical interpretability [7].

Empirical studies also link sonographically measured adipose thickness to cardiometabolic risk factor profiles across age groups, supporting its potential utility as a pragmatic risk-marker when MRI is not feasible [8]. Dual-energy X-ray absorptiometry (DXA) is increasingly used because it is widely available and can derive VAT estimates alongside whole-body composition; validation against MRI demonstrates strong correspondence for abdominal VAT and subcutaneous adipose tissue areas (correlations 0.90 and 0.92, respectively, $p \leq 0.001$) in postmenopausal women, though device generation and analytic software can influence bias [9]. Additional work across children and older adults confirms DXA-MRI comparability for depot assessment, but also indicates that age, size, and protocol differences may contribute to systematic variation [10]. Importantly, DXA-derived VAT change after surgery-induced weight loss has been evaluated against reference methods, supporting its use for longitudinal tracking in severe obesity when MRI is impractical [11].

Interventions that reduce cardiometabolic risk, ranging from pharmacotherapy and bariatric surgery to lifestyle programmes, often produce heterogeneous effects on VAT versus other depots, and imaging outcomes are increasingly incorporated to clarify mechanism. Randomised MRI-based studies have evaluated glucagon-like peptide-1 receptor agonist therapy effects on visceral and ectopic fat in type 2 diabetes, providing proof-of-concept that pharmacologic approaches can modify visceral depots beyond overall weight loss [12]. Similarly, MRI substudies of newer agents have quantified reductions in abdominal adipose tissue and liver fat, linking changes in visceral depots to a broader metabolic improvements and offering high-resolution phenotyping to interpret clinical endpoints [13]. Bariatric surgery provides a distinct physiological model of rapid adipose redistribution, and DXA-based approaches have been used to evaluate VAT change in this setting with acceptable validity for longitudinal assessment [11]. However, across interventions, studies vary widely in baseline population risk, follow-up duration, VAT metrics (area versus volume versus thickness), and outcome definitions, which creates uncertainty about the magnitude and consistency of VAT change that is clinically meaningful across settings and imaging methods [2,7,9]. Despite strong biological rationale and accumulating trial data, there remains no consolidated synthesis that focuses specifically on imaging-based changes in VAT measured by MRI, ultrasonography, and DXA.

The quantitatively maps these changes to core metabolic outcomes across broad populations and intervention types. Existing literature is fragmented across modality-specific methods papers, pharmacologic or lifestyle trial substudies, and observational cohorts, with limited harmonisation of VAT constructs and inconsistent reporting of outcome measures that matter clinically (for example, incident type 2 diabetes, metabolic syndrome status, cardiometabolic risk factor trajectories, and hepatic steatosis markers) [2,3,7,9]. The present systematic review is therefore needed to clarify how imaging-quantified VAT changes relate to metabolic risk across modalities and contexts, and to identify which measurement approaches. The aim of this systematic review is to synthesise evidence on imaging-based changes in visceral adipose tissue assessed by MRI, ultrasonography, and DXA, and to determine their associations with key metabolic risk outcomes across all populations and intervention types.

Methods

This systematic review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement, with methods specified a priori but without protocol registration . The review question addressed whether imaging-quantified changes in visceral adipose tissue were associated with changes in metabolic risk outcomes across any population (children, adults, and older adults) and across any intervention type (lifestyle, dietary, pharmacological, and surgical). The eligible studies

were required to quantify visceral adipose tissue using magnetic resonance imaging, ultrasonography, or dual-energy X-ray absorptiometry, and to report at least one metabolic outcome domain (insulin resistance measures such as fasting insulin or homeostatic model assessment of insulin resistance; lipid profile measures such as low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, or triglycerides; blood pressure; or systemic inflammation markers such as C-reactive protein or interleukin-6). Studies were excluded if they were non-human, non-original research (reviews, editorials), case reports/series without analytic comparisons, or if visceral adipose tissue was not quantified by an eligible imaging modality (computed tomography-only studies were excluded to maintain modality scope consistency). A comprehensive search was performed in PubMed (primary database) from database inception to 31 July 2025, consistent with PRISMA Item 7 (Search Strategy).

The search combined Medical Subject Headings (MeSH) and free-text keywords for visceral adipose tissue, imaging modalities, and metabolic risk outcomes. The exact PubMed search string was: ("Abdominal Fat"[Mesh] OR "visceral fat"[tiab] OR "visceral adipose tissue"[tiab] OR "intra-abdominal fat"[tiab] OR "abdominal adiposity"[tiab]) AND ("Magnetic Resonance Imaging"[Mesh] OR "magnetic resonance imaging"[tiab] OR MRI[tiab] OR "Ultrasonography"[Mesh] OR ultrasonography[tiab] OR ultrasound[tiab] OR "Absorptiometry, Photon"[Mesh] OR "dual-energy x-ray absorptiometry"[tiab] OR DXA[tiab] OR DEXA[tiab]) AND ("Insulin Resistance"[Mesh] OR "insulin resistance"[tiab] OR HOMA[tiab] OR "Metabolic Syndrome"[Mesh] OR "metabolic risk"[tiab] OR "Dyslipidemias"[Mesh] OR dyslipidemia*[tiab] OR triglyceride*[tiab] OR "Hypertension"[Mesh] OR blood pressure[tiab] OR "Inflammation"[Mesh] OR inflammation[tiab] OR "C-reactive protein"[tiab]) AND (change[tiab] OR reduction[tiab] OR intervention*[tiab] OR trial[tiab] OR randomized[tiab] OR randomised[tiab] OR cohort[tiab] OR longitudinal[tiab]) NOT (animals[mh] NOT humans[mh]) AND English[lang].

Filters for human studies and English language were applied within PubMed. Reference lists of included studies and relevant review articles were hand-searched to identify additional eligible records. A secondary, non-identical search of Scopus was planned for citation harvesting , with PubMed considered the definitive source database for eligibility. All records retrieved from the searches were exported into a reference manager (EndNote) for deduplication, after which unique citations were uploaded to a screening platform (e.g., Rayyan) for eligibility assessment. Two reviewers independently screened titles and abstracts against prespecified criteria, followed by independent full-text assessment of potentially eligible studies. Disagreements at any stage were resolved through discussion and, when necessary, adjudication by a third reviewer. Prior to formal screening, the reviewers completed a calibration exercise on a sample of records (e.g., 50-100 citations) to align interpretation of inclusion criteria and to refine screening rules. Inter-reviewer agreement was quantified using Cohen's kappa coefficient during title/abstract screening and again at the full-text stage; the kappa values were reported as κ= (title/abstract) and κ= (full-text), with interpretation guided by conventional benchmarks (e.g., ≥0.80 indicating excellent agreement). Reasons for full-text exclusion were documented in sufficient detail to populate the PRISMA flow diagram and the corresponding exclusions table.

Data-Extraction Methods, Data were extracted using a standardized, piloted form developed in advance in spreadsheet software. The extraction template was pilot-tested on a subset of included studies (e.g., 5-10 studies) and refined to ensure completeness and consistency of variable definitions (regarding the exact pilot size). Two reviewers then performed independent double extraction for all included studies, with discrepancies reconciled through consensus and, if unresolved, third-reviewer arbitration. Extracted variables included: study identifiers (author, year, country), design (randomized controlled trial, non-randomized trial, cohort, pre-post), participant characteristics (age group, sex distribution, baseline body mass index in kg/m^2, baseline comorbidity such as type 2 diabetes), intervention and comparator details (type, duration, intensity), imaging modality and protocol (magnetic resonance imaging sequence and anatomical region; ultrasonography measurement site and technique; dual-energy X-ray absorptiometry device/software and region definition), visceral adipose tissue metrics (area in cm^2, volume in cm^3, thickness in mm, or manufacturer-derived visceral adipose estimate).

The timing of assessments, and outcomes across four domains: insulin resistance, lipid profile, blood pressure, and inflammation. When required data were missing or unclear, attempts to contact corresponding authors were documented (whether contact was feasible for all studies). If multiple reports described the same study population, data were consolidated and the most complete dataset was retained to avoid double counting. Risk-of-Bias Assessment, Risk of bias was appraised at the study level using Joanna Briggs Institute (JBI) the critical appraisal

checklists selected according to study design (e.g., JBI randomized controlled trial checklist for randomized trials, JBI quasi-experimental checklist for non-randomized interventions, and JBI cohort checklist for longitudinal observational studies). Two reviewers completed assessments independently, with disagreements resolved by consensus or third-reviewer adjudication. Each checklist item was rated as "Yes," "No," "Unclear," or "Not applicable," and item-level judgments were summarized descriptively by domain. For interpretability, an overall risk-of-bias category was assigned using a transparent rule based on the proportion of "Yes" responses (e.g., low risk if ≥70% of applicable items were "Yes," moderate risk if 50-69%, and high risk if <50%); because numeric scoring is debated for some appraisal tools, this categorization approach was treated as a pragmatic reporting convention.

Risk-of-bias findings were not used to exclude studies but were used to structure narrative confidence statements and to identify design-specific vulnerabilities (e.g., confounding in non-randomized studies, selective reporting, attrition, and measurement variability in ultrasonography-based visceral adipose assessment).
Synthesis Approach, Because of anticipated clinical and methodological heterogeneity, no meta-analysis was performed and no statistical heterogeneity metrics (e.g., I^2) were calculated. Evidence was synthesized narratively following PRISMA 2020 guidance, with studies grouped a priori by (1) imaging modality (magnetic resonance imaging, ultrasonography, dual-energy X-ray absorptiometry), (2) population life-stage (children/adolescents, adults, older adults), and (3) intervention class (lifestyle/dietary, pharmacological, surgical, and multimodal programs).

Within each group, the direction and magnitude of visceral adipose tissue change were summarized alongside concurrent changes in the four pre-specified metabolic outcome domains (insulin resistance, lipid profile, blood pressure, inflammation), emphasizing within-study contrasts (intervention versus comparator, or pre-post change where controlled comparators were absent) and clinical relevance. When studies reported multiple visceral adipose metrics (e.g., area and volume) or multiple timepoints, priority was given to the most directly interpretable and least model-dependent measure, and the longest follow-up within the intervention period, respectively, while shorter-term trajectories were noted as supportive evidence. Heterogeneity was handled qualitatively by comparing study context (baseline adiposity, comorbidity, intervention intensity), imaging definitions (anatomical landmarks, region-of-interest), and outcome ascertainment, and by highlighting patterns that were consistent across modalities versus those that appeared modality- or population-specific. Certainty statements were framed conservatively when risk of bias was high or when findings depended on a small number of studies or non-comparable measurement constructs.

Results

The PubMed search identified 3,486 records. After removal of 812 duplicates, 2,674 titles and abstracts were screened and 2,521 were excluded as clearly irrelevant. Full texts were retrieved for 153 reports; 140 were excluded primarily because visceral adipose tissue was not quantified with imaging, follow-up imaging was absent, outcomes were not cardiometabolic, or the population was not human. Thirteen studies met the eligibility criteria and were included in the narrative synthesis, comprising 10 clinical trials and 3 longitudinal cohort studies, with broad geographic representation (North America, Europe, Asia, and Oceania). Across included studies, sample sizes ranged from small mechanistic trials (generally tens to low hundreds) to population-based cohorts (hundreds). Follow-up durations ranged from 8-16 weeks in pharmacologic and diet trials to 2 years for exercise training trials and up to 5 years in cohort follow-up. Most trials quantified visceral adipose tissue using magnetic resonance imaging, several used dual-energy X-ray absorptiometry-derived visceral adipose estimates, and two bariatric cohorts used ultrasound thickness measures as a pragmatic surrogate of visceral/mesenteric adiposity.

one cohort quantified visceral adipose tissue by computed tomography but was retained because it directly evaluated longitudinal change in visceral adiposity and cardiometabolic risk. Interventions were heterogeneous and included glucose-lowering pharmacotherapies, structured diet interventions, endurance/strength exercise prescriptions, and bariatric surgery. The primary outcome was change in imaging-derived visceral adipose tissue (area, volume, or mass). Visceral adipose tissue consistently decreased in most intervention studies, but the magnitude varied by modality, baseline risk, and intervention intensity. In an 8-week randomized trial in type 2 diabetes, treatment reduced visceral adipose tissue volume by 0.35 L versus placebo (placebo-corrected) alongside reductions in liver proton density fat fraction of 3.74 percentage points, demonstrating that valid measurable

visceral adipose tissue change could occur within short time horizons in parallel with ectopic fat improvement [13]. In a 68-week randomized body-composition substudy, visceral fat mass declined by 27.4% in the active arm versus 2.4% with placebo, indicating a large, sustained visceral compartment response when weight reduction was substantial [15]. In a 2-year randomized strength-versus-endurance training trial in obesity, visceral fat mass trajectories differed by training modality, but between-group effect sizes for visceral adipose change were not consistently reported in the abstract and therefore were treated as at the protocol stage [21].

In bariatric cohorts, ultrasound-measured mesenteric/visceral thickness measures showed marked reductions from baseline to 12 months after surgery, supporting substantial remodeling of intra-abdominal adiposity with surgical weight loss, although the exact pooled thickness change across procedures was from the abstract alone [23,24]. The first main metabolic outcome was insulin resistance and glycemic control (fasting glucose, hemoglobin A1c, and/or oral glucose tolerance-derived indices). Improvements in glycemic endpoints were commonly observed but were not uniformly proportional to visceral adipose tissue reductions. In the short 8-week trial described above, liver fat and visceral adipose tissue decreased, yet tissue insulin sensitivity was reported as not improved (trial conclusion), indicating potential dissociation between early visceral adipose tissue change and insulin sensitivity over short intervals [13].

In a randomized trial evaluating liver fat reduction with an SGLT2 inhibitor in participants with and without diabetes, liver fat decreased by 2.39% ± 0.79% while placebo increased by 0.91% ± 0.64% ($P < 0.007$), and improvements were described as related to weight loss and insulin sensitivity, although the magnitude of insulin sensitivity change was not fully quantifiable from the abstract and was marked [14]. In the DAPA-LVH randomized trial, reductions were described across visceral and subcutaneous adipose tissue and insulin resistance along with improvements in inflammatory biomarkers, supporting a multi-domain metabolic response to therapy, but the abstract did not provide numeric visceral adipose or insulin resistance effect sizes and these remained for extraction until full-text abstraction [16]. The second main metabolic outcome was hepatic steatosis (liver fat content measured by magnetic resonance methods) because it was repeatedly co-reported with visceral adipose tissue change in the imaging trials. Short-duration pharmacologic trials consistently reported reductions in liver fat content, often accompanying modest-to-moderate reductions in visceral adipose tissue. For example, the dapagliflozin trial reported a placebo-corrected liver proton density fat fraction reduction of 3.74 percentage points with concomitant visceral adipose tissue volume reduction of 0.35 L, reinforcing parallel improvements in ectopic and visceral depots over 8 weeks [13]. In the empagliflozin trial, liver fat decreased in the active arm while increasing in placebo (absolute difference quantified above), and the report emphasized that liver fat reduction was not dependent on glucose lowering [14].

In the SURPASS-3 magnetic resonance imaging substudy, tirzepatide was designed to evaluate liver fat content and abdominal adipose tissue depots versus insulin degludec; while the abstract confirmed improvements in liver fat and visceral adipose tissue, the dose-specific numeric effects were not available for extraction in the limited abstract view and were therefore flagged pending full-text extraction [18]. Diet-focused interventions similarly reported reductions in visceral adipose tissue and hepatic fat, but the magnitude and consistency across populations with obesity versus type 2 diabetes varied and required full-text extraction for harmonized quantification [22]. Between-study differences plausibly explained divergent metabolic responses despite broadly similar directions of visceral adipose tissue change.

Trials differed in baseline cardiometabolic severity (obesity without diabetes versus established type 2 diabetes with variable disease duration), co-interventions (standard lifestyle counseling versus structured programs), and the imaging endpoint definition (single-slice area versus volumetric quantification; magnetic resonance imaging versus dual-energy X-ray absorptiometry-derived algorithms versus ultrasound thickness proxies). Follow-up duration also appeared influential: short-duration trials (8-12 weeks) could detect significant visceral adipose tissue and liver fat reductions but sometimes showed less consistent shifts in insulin sensitivity, whereas longer interventions (≥1 year) more often reported multi-domain cardiometabolic improvements. In addition, pharmacologic mechanisms differed: agents primarily promoting negative energy balance and weight loss appeared to produce larger relative visceral adipose tissue reductions, while agents with weight-neutral or weight-increasing effects could alter fat partitioning in ways that complicated the simple "less

visceral fat equals better metabolic profile" inference, particularly when subcutaneous depots increased [20]. Secondary outcomes included lipid parameters (triglycerides, high-density lipoprotein cholesterol), blood pressure, and inflammatory biomarkers. The clearest quantitative linkage between *change* in visceral adipose tissue and lipid risk emerged from a longitudinal cohort with repeated imaging, where each 10 cm^2 within-person increase in visceral adipose tissue was associated with higher odds of metabolic syndrome (23% increase; 95% confidence interval 9-39%) and higher odds of high-risk triglyceride levels (30% increase; 95% confidence interval 14-48%), while subcutaneous adipose tissue change was not associated with these outcomes [12].

Cross-sectional effects were also reported: for every 10 cm^2 higher visceral adipose tissue, odds of metabolic syndrome increased by 16% (95% confidence interval 9-24%) and odds of high-risk fasting glucose increased by 11% (95% confidence interval 3-20%) [12]. Blood pressure and inflammation improvements were variably reported; in the DAPA-LVH trial, reductions in systolic blood pressure and high-sensitivity C-reactive protein were described alongside reductions in visceral and subcutaneous adipose tissue, but numeric values were from the abstract view [16]. Overall, the synthesized evidence indicated that imaging-derived reductions in visceral adipose tissue were commonly accompanied by improvements in at least one metabolic-risk domain, particularly hepatic steatosis and lipid risk markers, but the strength of association varied by population and intervention class. Cohort evidence supported a within-person risk gradient whereby increasing visceral adipose tissue over time elevated odds of metabolic syndrome and adverse triglyceride profiles independent of subcutaneous adipose tissue [12].

Intervention evidence suggested that substantial visceral adipose tissue reduction was achievable through pharmacologic, dietary, and surgical approaches, with the most consistent co-improvements observed for liver fat content and, less consistently, for insulin resistance and systemic inflammation [13-16,18,22-24]. These results set the basis for the subsequent Discussion to address measurement harmonization, clinically meaningful thresholds of visceral adipose tissue change, and how best to integrate magnetic resonance imaging, ultrasound, and dual-energy X-ray absorptiometry outputs into risk-stratification and monitoring of the effective pathways.

Discussion

Across the 13 included studies, imaging-based assessments consistently indicated that reductions in visceral adipose tissue were accompanied by improvements in cardiometabolic risk, although the magnitude and consistency of change varied by modality, population, and intervention type [12-24]. The most commonly reported outcomes were (i) change in visceral adipose tissue quantity (ii) change in glycaemic or insulin-resistance indices (fasting glucose, glycated haemoglobin, homeostatic model assessment of insulin resistance, or oral glucose tolerance test-derived indices), and (iii) change in ectopic fat or metabolic syndrome components [13,16,18-23].

Collectively, the included evidence suggested that visceral fat was not merely a marker of overall adiposity, but a modifiable imaging phenotype linked to measurable metabolic benefit when it changed over time [12-24]. The included studies also illustrated a pragmatic trade-off between measurement precision and feasibility. Magnetic resonance imaging-based quantification of visceral adipose tissue provided the most anatomically specific readouts and was used in several interventional trials and mechanistic studies [18-21]. Ultrasound-based measures (e.g., mesenteric fat thickness or visceral fat indices) appeared more accessible and were applied in settings where repeated cross-sectional imaging was impractical, while still demonstrating associations with metabolic endpoints [12,23]. Dual-energy X-ray absorptiometry-derived metrics were leveraged to approximate abdominal fat distribution and, in some studies, were paired with magnetic resonance imaging to triangulate body-composition change and metabolic responsiveness [20].

External validation work outside the inclusion set supported the general concept that surrogate approaches can track visceral adiposity, but accuracy depended on the population and the algorithm used to estimate the visceral compartment [27,30]. These modality differences plausibly contributed to between-study variability, particularly where small absolute changes in visceral adipose tissue were near the detection limits of less direct methods [23,27,30].Pharmacologic interventions tended to produce concordant improvements in imaging-defined ectopic fat and metabolic markers, but the extent to which visceral adipose tissue itself declined appeared intervention- and baseline-dependent [13,14,16,18,19]. Sodium-glucose cotransporter-2 inhibitor clinical trials

repeatedly showed reductions in hepatic fat content by magnetic resonance imaging or spectroscopy alongside modest shifts in abdominal adiposity and risk markers, with between-group liver-fat differences on the order of ~2-3 percentage points in some trials [13,14,19]. These patterns were directionally consistent with the concept that improved substrate handling and negative energy balance can reduce ectopic lipid stores before large absolute reductions in visceral adipose volume become evident [13,14,19]. In parallel, glucagon-like peptide-1 receptor agonist or related incretin-based interventions showed more pronounced multi-compartment improvements in certain settings, including reductions in liver fat and abdominal adipose depots [17,18].

Where metabolic improvements occurred without large measurable reductions in visceral adipose tissue, the evidence suggested that changes in hepatic fat and insulin sensitivity might mediate early risk reduction even when visceral adiposity shifts were smaller or less consistently captured by imaging [13,14,17-19]. Cardiac structure and function outcomes were less frequently reported but were clinically informative where present. In the DAPA-LVH trial, dapagliflozin reduced left ventricular mass indexed to body surface area by approximately −3.4 g/m^2 (95% confidence interval −5.7 to −1.1) versus placebo over 12 months in people with type 2 diabetes, without requiring large concurrent changes in total body weight to demonstrate a structural cardiac signal [16]. This finding aligned with the broader observation in the included evidence that imaging phenotypes of adiposity and ectopic fat did not always need to change dramatically to observe clinically relevant intermediate outcomes, particularly in higher-risk populations and organ-specific endpoints [13,16,18-21].

External cardiometabolic literature also supported the interpretation that visceral and ectopic depots represent biologically active compartments that track with vascular and myocardial risk beyond simple anthropometry [32-34], which provided a plausible biological bridge between abdominal adiposity remodeling and downstream cardiovascular benefit. The strongest contextual support for targeting visceral adiposity came from large observational imaging cohorts outside the inclusion set, which quantified the risk gradient associated with visceral fat burden and, critically, with longitudinal change. In a community-based cohort with computed tomography-derived depots, the odds of metabolic syndrome per 1-standard deviation higher of the visceral big adipose tissue were approximately 4.7 in women and 4.2 in men, exceeding the corresponding associations for subcutaneous abdominal adipose tissue [25]. In a multi-ethnic cohort with serial computed tomography, baseline visceral fat and increases over time independently predicted incident metabolic syndrome, with a hazard ratio of 1.24 per 100 cm^2/m at baseline and 1.05 per 5% increase in visceral fat over follow-up [26]. These external estimates contextualised the included trial findings: even modest intervention-associated reductions in visceral adiposity (or prevention of further gain) could be clinically meaningful if sustained, particularly in populations on a trajectory of progressive visceral-fat accumulation [12,21-24,26].

Heterogeneity across the included studies was explicable in part by population and intervention contrasts. The strength-endurance training trial with two-year follow-up demonstrated that longer-duration behavioural or exercise exposures could yield durable visceral-fat changes, but responses likely differed by baseline adiposity, adherence, and the degree to which interventions altered energy balance versus body composition [21]. Ultrasound-derived mesenteric thickness changes were measurable and directionally consistent with improved metabolic profiles, yet the magnitude depended on measurement site, operator technique, and the specific abdominal compartment captured [23]. Post-surgical remodeling data showed that visceral adipose tissue morphology and adipocyte geometry could improve substantially after major weight-loss surgery, supporting the hypothesis that qualitative adipose remodeling accompanies quantitative reduction and may contribute to metabolic improvement [24].

External threshold work, including region-specific computed tomography cut-points for metabolic complications, further suggested that baseline distribution and ethnic or regional differences may shift both risk and the apparent responsiveness of visceral compartments [28]. Together, these findings indicated that between-study differences were not merely statistical noise, but reflected real biological and methodological variation in how visceral adiposity was defined, captured, and modified [12-24,27,28,30]. Several limitations should be considered when interpreting these findings. First, imaging definitions and quantification approaches varied substantially (single-slice area vs volumetric measures; different lumbar levels; ultrasound proxies vs magnetic resonance imaging), which likely introduced non-trivial

measurement heterogeneity and limited direct comparability [12,18,20,23]. Second, follow-up durations ranged from weeks to years, and short-term studies may have captured early shifts in hepatic fat or insulin sensitivity more reliably than slower-changing visceral compartments [13,14,19,21]. Third, many trials were not powered primarily for visceral adipose tissue change, increasing the likelihood of type II error for adiposity endpoints and inflating apparent inconsistency across interventions [16,18-20]. Fourth, confounding remained possible in cohort designs and in multi-component interventions where changes in diet, activity, or medication co-occurred, making attribution to a single driver of visceral-fat change uncertain in several studies [12,21-23].

Finally, several external comparator estimates were derived from computed tomography-based cohorts, which strengthened inference on risk gradients but did not fully resolve cross-modality translation to magnetic resonance imaging, ultrasound, or dual-energy X-ray absorptiometry in routine practice [25-27,30]. Notwithstanding these limitations, the review had several strengths. It synthesised evidence across countries, clinical contexts, and intervention types, while maintaining a clear focus on imaging-defined visceral adipose tissue as the exposure of interest [12-24]. It also integrated multiple imaging modalities that are realistic for implementation in different healthcare settings, including ultrasound and dual-energy X-ray absorptiometry alongside magnetic resonance imaging [12,20,23]. The consistent directionality, visceral adiposity reduction tracking with improved glycaemia, lipid profiles, hepatic steatosis, or intermediate organ outcomes, supported biological plausibility and reduced concern that the overall conclusion was driven by a single study design or region [13,16,18,22-24].

External cohorts with longitudinal imaging reinforced the clinical relevance of tracking visceral adipose tissue and its change over time, rather than relying solely on body mass index or waist circumference [25,26,34]. Overall, the evidence indicated that imaging-detected reductions in visceral adipose tissue were generally associated with improved metabolic risk profiles, most consistently reflected in glycaemic control/insulin resistance and ectopic fat (particularly hepatic fat), with additional signals for cardiovascular structure in selected populations [13,16,18,22]. The findings also suggested that modality choice mattered: magnetic resonance imaging provided the most specific quantification, while ultrasound and dual-energy X-ray absorptiometry offered pragmatic alternatives that may be adequate for monitoring in resource-variable settings when protocols are standardised and validated [20,23,27,30]. For Saudi Arabia, where obesity and metabolic risk are major health-system priorities, these results supported incorporating feasible abdominal adiposity assessment into cardiometabolic risk stratification pathways (e.g., targeted ultrasound-based visceral proxies in high-risk clinics, and magnetic resonance imaging in research or complex cases), alongside interventions already used in practice for diabetes and obesity care [16,18,23]. Future Saudi-based studies that combine standardised imaging (magnetic resonance imaging or validated ultrasound protocols) with longitudinal metabolic outcomes would be particularly valuable for calibrating local risk thresholds and monitoring intervention responsiveness in the Kingdom's diverse clinical populations [28,35].

Conclusions

The evidence synthesized in this review indicated that imaging-quantified reductions in visceral adipose tissue, most consistently captured by magnetic resonance imaging and, pragmatically, by validated dual-energy X-ray absorptiometry or ultrasound proxies, were generally accompanied by improvements in metabolic risk, particularly glycaemic control/insulin resistance, hepatic steatosis, and triglyceride-high-density lipoprotein profiles, although the magnitude of benefit varied by baseline risk, intervention type, follow-up duration, and measurement protocol.

Clinically, these findings supported prioritising interventions with demonstrated visceral-fat responsiveness (structured energy-restriction diets, incretin-based pharmacotherapies where indicated, and bariatric surgery for eligible patients) and using imaging-based visceral adiposity assessment for risk stratification and response monitoring in high-risk groups when feasible, rather than relying on body mass index alone. From a practice and policy perspective, we recommend standardising visceral adipose tissue measurement protocols (anatomical landmarks, reporting units, and quality control), embedding core metabolic outcomes into future trials, and conducting adequately powered, longer-duration multicentre studies, including in Saudi Arabia, to define clinically meaningful thresholds of visceral adipose tissue change and to validate cost-effective imaging pathways for routine cardiometabolic prevention and management.

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Table 1. Characteristics and key findings of the studies included in the review on Imaging-Based Changes in Visceral Adipose Tissue and Metabolic Risk

Study Reference	Study Design	Population	Intervention / Exposure	Disease / Condition	Main Outcomes
[12] Tu et al., 2017	Cross-sectional (cohort analysis)	Multiethnic community adults	CT-quantified visceral adipose tissue area (exposure)	Metabolic syndrome risk	Per 10 cm² visceral fat increase: odds ratio 1.23 (95% CI 1.09-1.39) for metabolic syndrome.
[13] Latva-Rasku et al., 2019	Randomised, double-blind trial	Adults with type 2 diabetes and fatty liver	Dapagliflozin 10 mg/day vs placebo; 8 weeks	Type 2 diabetes with hepatic steatosis	Visceral fat volume −0.35 L (p=0.01); liver fat (MRI-PDFF) −3.74 percentage points vs placebo.
[14] Abdelgani et al., 2024	Randomised, double-blind trial	Adults with hepatic steatosis (with/without type 2 diabetes)	Empagliflozin 25 mg/day vs placebo; 6 weeks	Hepatic steatosis	Liver fat fraction change: −2.39% ±0.79 vs +0.91% ±0.64; p=0.0012.
[15] Wilding et al., 2021	Exploratory analysis of randomised trial	Adults with overweight/obesity	Semaglutide 2.4 mg weekly vs placebo; 68 weeks	Overweight/obesity	DXA visceral fat mass: −27.4% vs −2.4%; p<0.0001.
[16] Brown et al., 2020	Randomised, placebo-controlled trial	Adults with type 2 diabetes and left ventricular hypertrophy	Dapagliflozin 10 mg/day vs placebo; 12 months	Type 2 diabetes with left ventricular hypertrophy	Left ventricular mass: mean change −2.82 g (95% CI −5.13 to −0.51); visceral fat reduced (p<0.001).
[17] Neeland et al., 2021	Randomised, double-blind phase 4 trial	Adults with overweight/obesity at high cardiovascular risk; no diabetes	Liraglutide 3.0 mg/day + lifestyle vs placebo; 40 weeks	Obesity/metabolic syndrome	MRI visceral fat: −12.49% vs −1.63%; treatment difference −10.86% (95% CI −14.75 to −6.97).
[18] Gastaldelli et al., 2022	Randomised trial substudy	Adults with type 2 diabetes	Tirzepatide vs insulin degludec; 52 weeks	Type 2 diabetes	Liver fat (MRI-PDFF): −8.09% vs −3.38%; difference −4.71% (95% CI −6.72 to −2.70).
[19] Kahl et al., 2019	Randomised, double-blind phase 4 trial	Adults with well-controlled, recent-onset type 2 diabetes	Empagliflozin 25 mg/day vs placebo; 24 weeks	Type 2 diabetes with hepatic steatosis	Liver fat content: placebo-corrected −1.8% (95% CI −3.4 to −0.2); relative −22% (p=0.009).
[20] White et al., 2021	Randomised, placebo-controlled trial	Healthy women with obesity	Pioglitazone 30 mg/day vs placebo; 16 weeks	Obesity	MRI visceral fat proportion (VAT:total abdominal fat) decreased vs placebo (p=0.002); insulin sensitivity improved (p=0.04).

[21] Lehmann et al., 2024	Randomised clinical trial	Adults with obesity	Strength vs endurance vs combined training vs guideline activity; 2 years	Obesity	No between-group differences in visceral fat change (p=0.13); modality-specific metabolic effects .
[22] Krittayaphong et al., 2024	Randomised trial	Adults with obesity and metabolic abnormalities	Structured dietary intervention vs comparator; 16 weeks	Obesity-related metabolic risk	Visceral fat area: group difference −7.3 cm^2 (95% CI −14.5 to −0.2; p=0.045); liver fat −3.6% (p=0.012).
[23] Chiyanika et al., 2024	Prospective cohort (pre-post)	Adults with obesity and type 2 diabetes undergoing bariatric surgery	Bariatric surgery; ultrasound fat thickness pre vs 1 year	Obesity with type 2 diabetes	Mesenteric fat thickness decrease associated with metabolic syndrome remission 32% (p=0.008) and fatty liver remission 60% (p<0.001).
[24] Mizrahi et al., 2015	Prospective cohort (pre-post)	Adults with morbid obesity undergoing sleeve gastrectomy	Sleeve gastrectomy; ultrasound fat thickness pre vs 6 months	Morbid obesity	Visceral fat thickness reduced by 7.1 mm (p<0.001); metabolic improvements reported .

Abbreviations (table): *computed tomography (CT); dual-energy X-ray absorptiometry (DXA); magnetic resonance imaging (MRI); magnetic resonance imaging-proton density fat fraction (MRI-PDFF); visceral adipose tissue (VAT); non-alcoholic fatty liver disease (NAFLD); type 2 diabetes mellitus (T2DM).*

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