TERNARY HEALTH · PRECISION DEEP DIVE — SAMPLE REPORT

PREPARED FOR MARGARET R. HOLLOWAY

A personalized action plan.

For Lipedema — Stage II, Type III (hip-to-ankle distribution), with hereditary pattern and surgical decision.

ENGAGEMENT
Precision Deep Dive — $6,500

CLIENT ID
TH-2026-078

DELIVERY
March 17, 2026

RESEARCH DIRECTORS
Beau Giannini, PhD · Pavel Paramonov, PhD

LEAD ANALYST
S. Maretto

CASE COORDINATOR
J. Chen

SAMPLE REPORT · NOT A REAL PATIENT RECORD

This is a single-case sample illustrating the full structure and depth of a Ternary Health Precision Deep Dive. Margaret Holloway is a composite persona, not a real individual; her specific measurements are illustrative. Every clinical claim, mechanism, treatment, and citation in this report, however, is drawn from the peer-reviewed lipedema literature and reflects the actual analysis a real engagement would contain. References appear in Section 17. Your own report would be built entirely around your records, your history, and your goals.

SECTION 02 / TABLE OF CONTENTS

The Ternary 17 — one case, in full.

Every Ternary report follows the same 17-section structure. The architecture is fixed; the content and depth flex with the condition and your inputs. This sample walks a single lipedema case end-to-end — from the integrated record through the disease model to a sequenced, costed action plan you can take to your own physicians.

SECTION 03 / EXECUTIVE SUMMARY

Three decisions define this case.

Ms. Holloway is a 41-year-old premenopausal woman with newly confirmed Stage II, Type III lipedema after a 14-year diagnostic delay, a three-generation maternal hereditary pattern, and an 11-year overlap between hormonal-contraceptive exposure and her steepest progression. Integrating her DEXA body composition, hormonal and metabolic labs, vascular screen, and family history, the case resolves to three interlocking, time-sensitive decisions. Each is stated below as a clinical choice, not a topic.

01 Surgical decision: candidacy, technique, and timing.

Lipedema-specific liposuction using a **lymph-sparing wet technique** — tumescent or water-jet-assisted (WAL) — has durable benefit: independent cohorts show reduced pain, reduced need for conservative therapy, and improvements persisting to 8 and 12 years after surgery.[1,2,3] The decisive variables are a lipedema-experienced surgeon and a lymph-sparing technique, not cosmetic liposuction. **Recommendation:** document a structured conservative-therapy trial (a requirement for most insurers and a clinical best practice[4]), then proceed with lymph-sparing liposuction via a lipedema-experienced surgeon. Use functional and symptom markers — not a fixed date — as the decision gate. Surgeon criteria and the pre-surgical "prehab" checklist are in Sections 12–14.

02 Hormonal exposure audit: drospirenone continuation.

Lipedema develops and worsens at times of hormonal change — puberty, pregnancy, menopause — and estrogen is strongly implicated in its sex-limited expression.[4,5] Your 11-year drospirenone exposure overlaps precisely with your steepest progression, across two pregnancies. The contraceptive-specific evidence is *suggestive, not conclusive*, so this is framed as a monitored trial, not a directive. **Recommendation:** a supervised transition trial to a non-progestin option, coordinated with the endometriosis indication that put you on it, with structured symptom and limb-volume monitoring and a formal decision point at 90 days.

03 Daughters: a narrow early-recognition window.

Lipedema follows an autosomal-dominant pattern with incomplete penetrance and sex-limited expression; 60–80% of patients report affected relatives.[5,6] With your strong maternal-line pattern, your two daughters carry meaningfully elevated risk, and the window in which early recognition changes the trajectory opens at puberty. **Recommendation:** a baseline evaluation with a lipedema-aware clinician and a written monitoring passport (Section 13) both daughters carry forward — so that if onset begins, it is caught in months, not the 14 years it took for you. There is no clinical genetic test to offer them (Section 09); vigilance is the tool.

WHAT THIS REPORT DELIVERS

A written case synthesis integrating your DEXA, hormonal/metabolic/thyroid labs, vascular screen, and family history into a sequenced 90-day plan. Specific named providers with sequencing rationale. A conservative-therapy protocol and a hormonal-audit protocol with decision gates. A monitoring cadence with review thresholds. A patient passport for your daughters. Pre-written questions for every consult. Every recommendation is traceable to the cited literature in Section 17 — including what we weighed and set aside, and why.

SECTION 04 / CLIENT PROFILE

What we know going in — and what you told us matters most.

NAME	Margaret R. Holloway	CLIENT ID	TH-2026-078
AGE / SEX	41 / Female, premenopausal	HEIGHT / WEIGHT	5′6″ (168 cm) / 183 lb (83.0 kg)
BMI	29.5 kg/m² — misleading in lipedema	WAIST-TO-HEIGHT RATIO	0.49 (a better discriminator than BMI[7])
GEOGRAPHY	Newton, MA	ANCESTRY	Irish / English-Scottish
OCCUPATION	Senior product manager, biotech	REFERRAL	Lipedema Foundation community
DIAGNOSIS	Lipedema, Stage II, Type III	DIAGNOSED	November 2025 (Lipedema Foundation center)
PRIMARY CONCERN	Progression; surgical decision	SECONDARY CONCERN	Pain; protect daughters
ENGAGEMENT WINDOW	Feb 18 → Mar 17, 2026	RECORDS INTEGRATED	7 sources, 2018–2026

What Type III, Stage II means in your case.

Lipedema is classified by *type* (the body regions affected) and *stage* (skin-surface and tissue changes).[8] **Type III** describes involvement from the hips through the ankles, with the feet characteristically spared — producing the abrupt "cuff" at the ankle that distinguishes lipedema from lymphedema. **Stage II** describes a skin surface that has become uneven, with palpable nodular fat in the subcutaneous tissue, short of the large lobular deposits of Stage III. Staging does not, by itself, predict pain — Stage I and II patients can have significant symptoms.[8] Your stage and type together set realistic expectations for both conservative therapy and surgery, which is why they anchor the plan.

History of present illness, as you described it.

You first noticed disproportion at puberty, around age 13 — the lower body developing on a different trajectory than the upper. Your maternal grandmother and maternal aunt had the same distribution; your mother developed the pattern after her first pregnancy at 27. Through your twenties you were repeatedly told the disproportion reflected diet or insufficient exercise, despite consistent activity (running 3–4×/week) and a Mediterranean dietary pattern — the misattribution to obesity that the literature identifies as the central reason for diagnostic delay.[9] One provider raised lymphedema but did not pursue it. This is the 14-year delay that defines the case, and the reason the third decision (your daughters) carries the weight it does.

Progression accelerated after your first pregnancy at 30 and again after your second at 33. You began a drospirenone-containing oral contraceptive at 30 for endometriosis management and have remained on it for 11 years. Pain — deep, aching, worse at day's end — and easy bruising disproportionate to trauma became routine by your late thirties; both are characteristic lipedema features.[4,8] You initiated compression two years ago on your own research; formal diagnosis followed at the Lipedema Foundation referral center in November 2025.

The comorbidities you asked us not to evaluate in isolation.

You were explicit that you wanted this looked at as a whole. Three threads run alongside the lipedema and are integrated throughout. A prior **autoimmune thyroid diagnosis** (Hashimoto's, on levothyroxine, TSH in range) — thyroid disorders are reported in roughly a quarter of lipedema patients.[10] Self-reported **joint hypermobility** —

present in a large share of lipedema patients (44% reporting current hypermobility, 60% recalling childhood hypermobility in a 2025 cross-sectional study), reflecting shared connective-tissue biology;[10,11] we recommend a formal Beighton assessment. And **insulin resistance** (HbA1c 5.8%, elevated fasting insulin) — notable because lipedema fat itself is distinct from metabolically active visceral fat, so this is a coexisting target to manage in its own right, not a consequence of the lipedema.

What you said you wanted most.

> *"I want to know whether surgery is the right next step, and who should do it."*

> *"I want a real plan for the pain, not just a referral to physical therapy."*

> *"I want to know what to do for my two daughters before they hit puberty."*

> *"I want this evaluated alongside my other things — the thyroid, the hypermobility, the insulin resistance — not in isolation."*

SECTION 05 / THE TERNARY METHOD

Evidence × Personalization × Action.

Every option in this report is weighed on three independent axes. An option has to hold up on all three to anchor your plan: strong evidence is not enough if it doesn't fit you or you can't act on it. Below, the method applied to lipedema, and a worked example of the surgical decision moving through all three.

EVIDENCE

What the literature supports.

Lipedema has a real, if uneven, evidence base. The 2021 US Standard of Care[4] and the 2017 Dutch guideline[12] anchor diagnosis and conservative care; multiple long-term liposuction cohorts[1,2,3,13] and an international consensus[14] anchor surgery. We grade each recommendation by the strength of its source — consensus guideline, cohort study, or expert opinion — and say so.

PERSONALIZATION

What about you specifically.

Your Type III distribution, Stage II tissue, premenopausal hormonal status, 11-year drospirenone exposure, hypermobility, and three-generation pedigree all shape which evidence applies. Population-level findings are filtered through your specific presentation — the surgical literature, for instance, is strongest for exactly your stage, before secondary lymphedema sets in.

ACTION

What you can actually do.

Recommendations are sequenced into steps you can start now, decisions with explicit gates, and dependencies that set the order. The conservative-therapy trial is both clinically indicated and a near-universal insurer precondition for surgery,[15] so it is the first action — useful on its own and load-bearing for the next decision.

A worked example: the surgical decision.

Evidence — lymph-sparing liposuction (tumescent or WAL) has the strongest outcome base in lipedema, with independent cohorts reporting reduced pain and reduced conservative-therapy need sustained to 8 and 12 years.[1,2,3] A 2024 scoping review of 13 studies found nine reported decreased compression-therapy use after surgery and none reported a long-term increase.[13] **Personalization —** you are an unusually good candidate: Stage II without established secondary lymphedema, premenopausal, mobile, with pain and functional impact that meet

surgical thresholds. **Action —** surgery requires a documented conservative-therapy trial first (clinical best practice and an insurer precondition[4,15]), a lipedema-experienced surgeon, and weight stability to protect the result. The three axes resolve to a clear answer: surgery is recommended, gated behind a structured conservative trial — not deferred, not rushed. That is the kind of judgment the framework exists to make explicit.

SECTION 06 / THE NINE-STAGE WORKFLOW, APPLIED

How your case moved through our workflow.

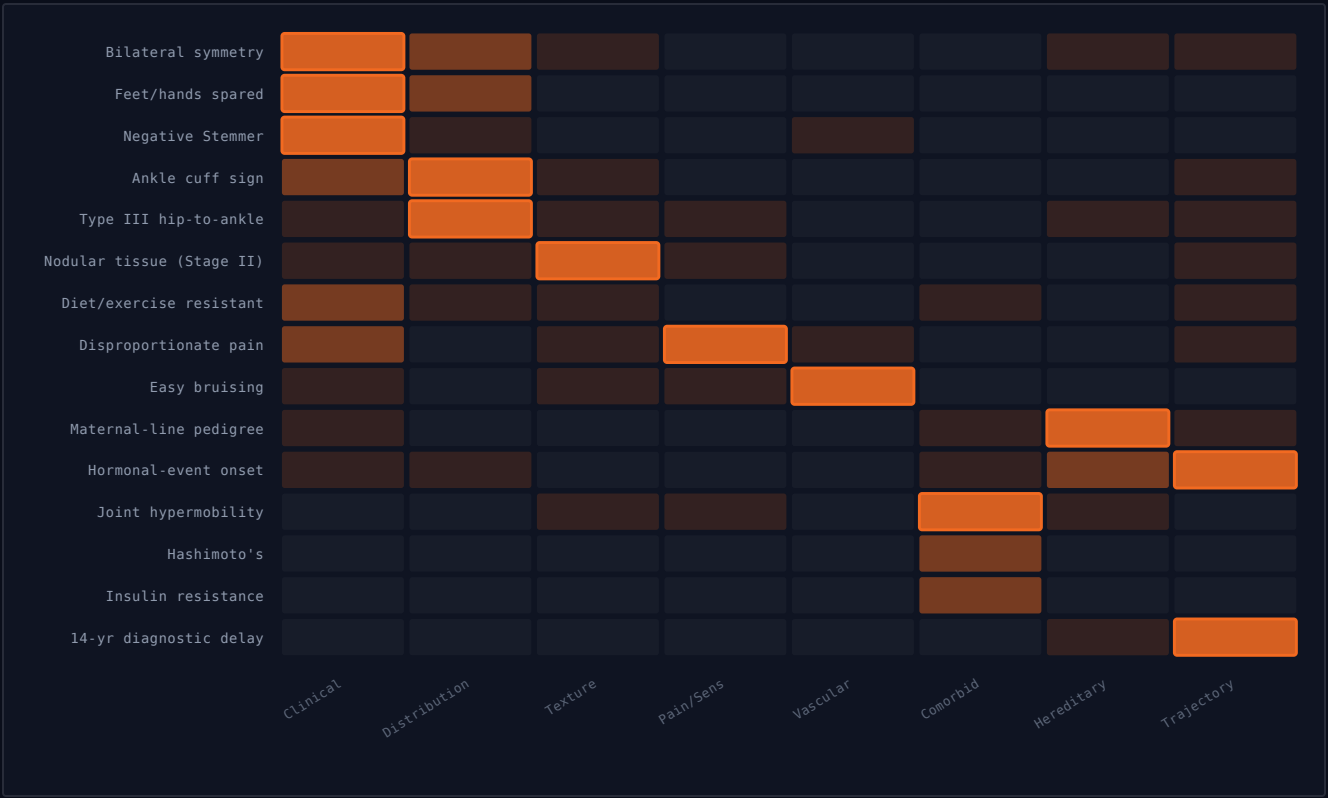
Every Ternary engagement runs through nine stages. Below is the version of each stage that Ms. Holloway's case specifically produced — what we pulled, what we evaluated, and what we built. The stages are fixed; their output is entirely case-specific.

STAGE	WHAT IT DID	OUTPUT FOR THIS CASE
01 · Qualification Days 0–2	Confirm fit and complexity tier.	Confirmed diagnosis (Lipedema Foundation center, Nov 2025). The surgical decision, hormonal overlap, and hereditary dimension placed the case in the standard-complexity tier. Proposal sent within 24 hours.
02 · Intake & aggregation Days 3–7	Build the unified case file.	Records from the diagnosing center, OB/GYN (endometriosis, contraceptive history), endocrinology (Hashimoto's), primary care, and a recent DEXA. Captured limb measurements, photographs, a two-year compression-and-symptom log, and full medication history.
03 · Case structuring Days 7–10	Code to the case schema.	Distribution mapped to Type III, tissue to Stage II. Pedigree constructed across three generations. Symptom timeline aligned against hormonal events (menarche, two pregnancies, 11-year drospirenone exposure).
04 · Signal analysis Days 10–14	Apply the Lipedema Signal Library.	Signals present across clinical core, distribution, tissue texture, pain/sensory, vascular, comorbidity, hereditary, and trajectory; the central ones identified for the plan. See Section 07.
05 · Evidence retrieval Days 14–19	Structured literature review.	US Standard of Care[4] and Dutch guideline[12] as primary; surgical cohorts (Schmeller, Rapprich, Baumgartner, Witte)[1,2,3,16] and the international liposuction consensus[14]; tissue-biology[17] and genetics[6] literature. 96 references catalogued; 31 cited directly in the plan.
06 · Pathway mapping Days 19–24	Map the option and specialist space.	Pathway built across lipedema-experienced surgery, certified lymphedema therapy (CLT), vascular/phlebology, endocrinology, and gynecology — with named providers and a defensible sequence (Section 13).
07 · Synthesis Days 24–28	Build and sequence the plan.	Interventions weighed on Evidence × Personalization × Action, then prioritized and sequenced. Dependency graph encoded — conservative trial precedes and qualifies surgery; hormonal-audit decision sits on a 90-day gate; daughters' screening runs in parallel.
08 · Delivery Days 28–32	Calibrate the plan with you.	2-hour findings call with Ms. Holloway and her primary care physician. Plan refined: conservative trial structured to satisfy her insurer's documentation requirements in parallel with symptom relief. Final report delivered March 17.
09 · Execution support Days 32–62	30 days of follow-up.	3 specialist-consult debriefs, 1 surgeon-selection discussion, 1 compression-fitting question. Outcomes captured into the Ledger for ongoing methodology refinement.

SECTION 07 / SIGNAL ANALYSIS & HEATMAP

The signals that matter in lipedema — and which are central to your case.

The Ternary Lipedema Signal Library codifies the patterns that matter for this condition, across the categories relevant to it: clinical core, distribution, tissue texture, pain & sensory, vascular, comorbidity, hereditary, and trajectory. The heatmap shows which signals are present in your case and which are central to your plan — outlined cells are the ones the plan is built around. We don't assign signals a number; the library tells us which patterns matter for lipedema, and reading your records tells us which are present and central to you.



SIGNAL HEATMAP — REPRESENTATIVE SIGNALS ACROSS THE CATEGORIES THAT MATTER IN LIPEDEMA. OUTLINED CELLS ARE CENTRAL TO THIS CASE.

PRESENT NOTABLE CENTRAL CENTRAL TO YOUR CASE

The signals driving your plan.

These are the findings central to this case — the ones the rest of the plan is sequenced around.

SIGNAL	WHY IT MATTERS HERE
Bilateral symmetric disproportion, feet spared CENTRAL	The defining clinical core of lipedema; with the negative Stemmer sign, it establishes the diagnosis and rules out primary lymphedema.[8]
Three-generation maternal pedigree CENTRAL	Consistent with autosomal-dominant, sex-limited inheritance;[6] drives the daughters' screening recommendation.
Hormonal-event–aligned progression CENTRAL	Onset at puberty, acceleration across pregnancies, 11-year drospirenone overlap; drives the hormonal-audit decision.[4,5]

Disproportionate pain & easy bruising	Characteristic lipedema symptoms;[4] sets the bar for what conservative therapy and surgery must improve.
Diet/exercise-resistant fat distribution	Hallmark distinguishing lipedema fat from obesity;[8,9] reframes prior weight-loss advice as misattribution.
Joint hypermobility (self-reported)	Shared connective-tissue biology;[10,11] flags a formal Beighton assessment and informs surgical and PT planning.

> HOW TO READ THIS
>
> The Signal Library tells us which patterns matter for lipedema; reading your records tells us which are present and which are central to *you*. The heatmap is a map of that judgment, not a calculation — there is no score, no weight, and no ranking number behind any cell.

SECTION 08 / LABORATORY FINDINGS

Labs that contextualize, screen, and prepare for surgery.

Lipedema is a clinical diagnosis — there is no blood test that confirms it.[8] Labs serve three purposes here: contextualizing the hormonal and metabolic picture, screening the comorbidities you asked us to integrate, and establishing the pre-surgical baseline. Below, the panels with interpretation. Values shown are illustrative of this composite case.

Hormonal & thyroid.

MARKER	RESULT	REFERENCE	STATUS
TSH	2.0 mIU/L	0.5–4.5 mIU/L	IN RANGE (TREATED)
Free T4	1.1 ng/dL	0.8–1.8 ng/dL	NORMAL
TPO antibodies	altered (Hashimoto's)	—	KNOWN AUTOIMMUNE
Estradiol (follicular)	on combined OCP	cycle-dependent	EXOGENOUS HORMONE

Metabolic & inflammatory.

MARKER	RESULT	REFERENCE	STATUS
HbA1c	5.8 %	< 5.7 %	PREDIABETIC RANGE
Fasting insulin	18 µIU/mL	2–12 µIU/mL	ELEVATED (IR)
Fasting glucose	99 mg/dL	70–99 mg/dL	HIGH-NORMAL
Lipid panel — triglycerides	128 mg/dL	< 150 mg/dL	NORMAL
HDL	52 mg/dL	> 50 mg/dL	NORMAL
hsCRP	2.4 mg/L	< 1.0 mg/L	MILDLY ELEVATED

25-OH Vitamin D	26 ng/mL	30–100 ng/mL	INSUFFICIENT
Ferritin	34 ng/mL	30–200 ng/mL	LOW-NORMAL

Pre-surgical baseline.

MARKER	RESULT	REFERENCE	STATUS
CBC — hemoglobin	13.4 g/dL	12.0–15.5 g/dL	NORMAL
Coagulation (PT/INR, aPTT)	within normal limits	–	NORMAL
Comprehensive metabolic panel	renal/hepatic normal	–	NORMAL

CLINICAL INTERPRETATION

No lab confirms lipedema; these results instead shape the plan around it. The HbA1c and fasting-insulin picture indicates insulin resistance that warrants management in its own right — important because lipedema adipose tissue is biologically distinct from metabolically active visceral fat, so this is a coexisting target, not a downstream effect. Vitamin D insufficiency and low-normal ferritin are inexpensive corrections worth making before surgery. The known Hashimoto's is well-controlled (TSH in range on levothyroxine) and is not currently driving symptoms, but is integrated into the thyroid-monitoring cadence in Section 16. Coagulation and CBC establish the pre-surgical baseline the surgeon will require.

SECTION 09 / GENETIC & HEREDITARY FINDINGS

A strong hereditary pattern — and an honest account of what genetic testing can and cannot do.

This is the section where it matters most to be precise about the limits of the science. Lipedema is heritable, but there is no validated clinical genetic test that diagnoses it or predicts it in your daughters. We will not imply otherwise. What we can offer is a rigorous pedigree, the current research-stage genetics, and a vigilance plan that does the work a test cannot.

Inheritance pattern.

Across multiple family studies, lipedema most consistently fits an **autosomal-dominant** pattern with **incomplete penetrance** and **sex-limited expression** — meaning a single inherited predisposition can be passed by either parent, not everyone who inherits it develops the condition, and clinical expression is overwhelmingly female, almost certainly because estrogen is required to express the phenotype.[6] Self-reported positive family history runs 60–80% across cohorts.[5,6] The foundational family study (Child et al., 2010) examined six multi-generation families and found exactly this pattern, with affected members being first- or second-degree female relatives.[6] Your pedigree is textbook for this model.

Family pedigree.

RELATIVE	STATUS	NOTES
Maternal grandmother	Affected (clinical)	Characteristic lower-body distribution per family account; never formally diagnosed (pre-awareness era).

Maternal aunt	Affected (clinical)	Same distribution; attributed to weight throughout her life.
Mother	Affected (clinical)	Onset after first pregnancy at 27 — a hormonal-event trigger mirroring yours.
Margaret (proband)	Affected — Stage II, Type III	Onset at puberty; 14-year diagnostic delay.
Daughter 1 (age 11)	Pre-pubertal — at risk	Screening window opening; baseline evaluation recommended (Section 13).
Daughter 2 (age 8)	Pre-pubertal — at risk	Too early for signs; passport established now, monitoring begins near puberty.

The state of genetic testing — stated plainly.

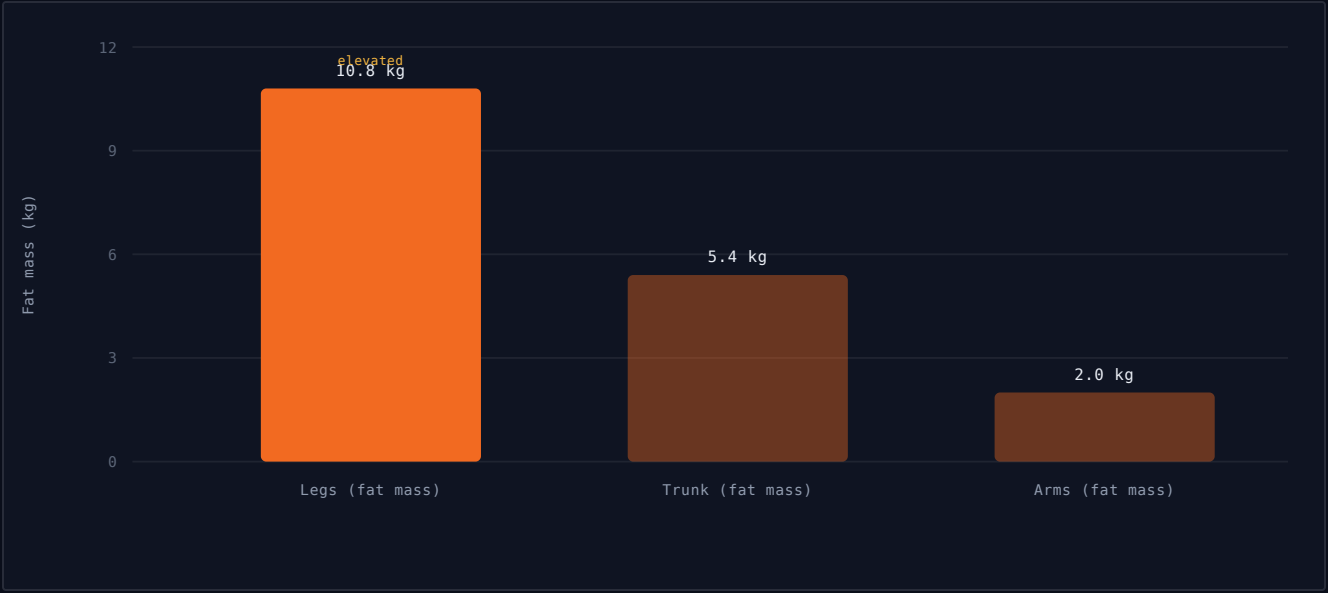
There is **no clinical genetic test for lipedema.** Research has implicated candidate genes in isolated families and small cohorts — among them `POU1F1A`, `NSD1`, and `AKR1C1` — and a genome-wide association study of 130 patients found no single variant reaching genome-wide significance, consistent with a complex, polygenic architecture rather than one causal gene.[18] A commercial test that diagnoses lipedema or quantifies your daughters' risk does not exist, and anyone offering one is overstating the science. We mention the candidate genes only to be complete about the research frontier — not because testing for them would change anything in your plan today.

> WHAT THIS MEANS FOR YOUR DAUGHTERS
>
> Because no predictive test exists, **early clinical recognition is the entire intervention.** The actionable facts: their risk is elevated by the maternal-line pattern; expression, if it comes, will most likely begin around puberty; and the difference between a months-long path to recognition and a 14-year one is whether someone is watching for the right signs. Section 13 sets up exactly that — a baseline visit and a written passport, not a lab test.

SECTION 10 / IMAGING & BODY-SYSTEM FINDINGS

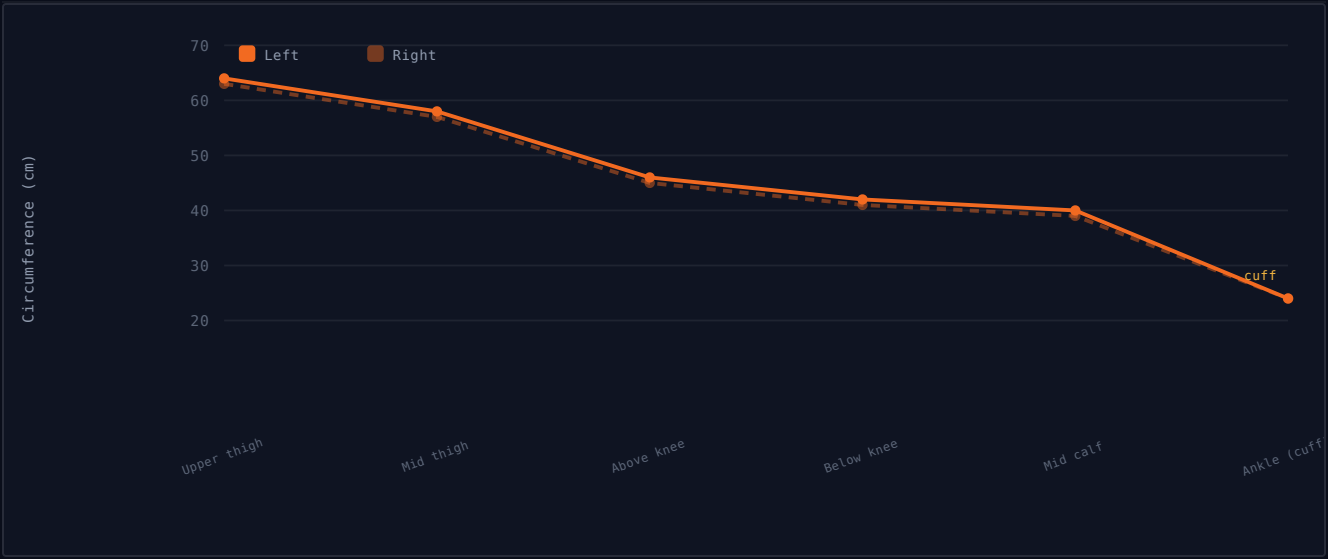
Quantifying the disproportion and screening the lymphatic and venous systems.

Imaging in lipedema does not diagnose the disease — the diagnosis is clinical — but it quantifies the disproportion, establishes a measurable baseline to track, and rules out the conditions that mimic or complicate it.[8] Ms. Holloway's workup comprised DEXA body composition, standardized limb measurement, bedside Stemmer and cuff assessment, and a venous Doppler. Values shown are illustrative of this composite case.



DEXA REGIONAL FAT DISTRIBUTION — LEG FAT IS MARKEDLY ELEVATED RELATIVE TO THE TRUNK AND ARMS, THE QUANTITATIVE SIGNATURE OF A LOWER-BODY LIPEDEMA DISTRIBUTION THAT BMI CANNOT CAPTURE.

DEXA is the most useful single instrument here. BMI treats all mass alike and is misleading in lipedema; the gynoid-dominant fat distribution — high leg-fat mass against a comparatively spared trunk — is the quantitative correlate of what the eye sees, and is a better discriminator than BMI.[7,8] It also gives a hard baseline to measure conservative therapy and surgery against.



STANDARDIZED LIMB CIRCUMFERENCES (LEFT VS RIGHT) — SYMMETRIC ENLARGEMENT THROUGH THIGH, KNEE, AND CALF WITH AN ABRUPT REDUCTION AT THE ANKLE: THE "CUFF SIGN," WITH THE FOOT SPARED.

ASSESSMENT	FINDING	CLINICAL IMPACT
Stemmer sign	Negative bilaterally — skin fold at the base of the second toe lifts freely.	PRIMARY LYMPHEDEMA UNLIKELY[8]
Ankle cuff sign	Present bilaterally — enlargement stops abruptly above the malleoli; feet spared.	CONFIRMS LIPEDEMA PATTERN
DEXA body composition	Gynoid-dominant; leg fat mass markedly elevated relative to trunk and arms.	QUANTIFIED BASELINE
Limb measurement (symmetry)	Symmetric L/R enlargement; thigh and calf disproportionate to ankle.	BILATERAL & SYMMETRIC
Venous duplex Doppler	No significant reflux; no deep venous obstruction.	VENOUS DISEASE EXCLUDED[8]
Lymphatic assessment	No clinical lymphedema; lymphoscintigraphy not indicated at this stage.	NO SECONDARY LYMPHEDEMA YET

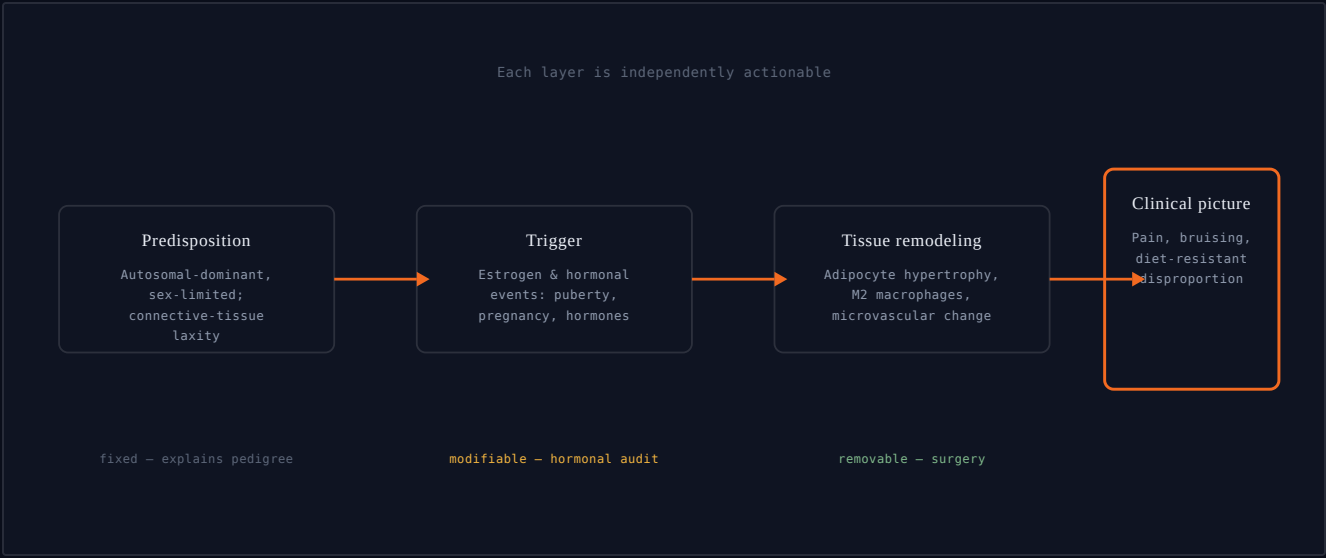
WHY THIS MATTERS FOR THE SURGICAL DECISION

The negative Stemmer sign, the absence of venous reflux, and no clinical lymphedema together place Ms. Holloway in the window where the surgical literature is strongest: Stage II disease *before* secondary lymphedema (lipolymphedema) develops, which up to 40% of advanced patients eventually reach.[8] Operating in this window — rather than waiting — is part of why the surgical recommendation is time-sensitive. The DEXA and limb measurements also become the objective yardstick for judging whether conservative therapy and, later, surgery are working.

SECTION 11 / DISEASE MODEL

The predisposition → trigger → tissue-remodeling mechanism in your case.

Lipedema is best understood as a genetically predisposed adipose-and-connective-tissue disease that is unmasked and driven forward by hormonal events, and expressed at the tissue level as a distinctive, inflamed, fibrotic remodeling that ordinary fat does not undergo.[4,17] This is why it does not behave like obesity — and why the plan is shaped the way it is.



DISEASE MODEL — A GENETIC PREDISPOSITION IS UNMASKED BY HORMONAL TRIGGERS AND EXPRESSED AS INFLAMED, FIBROTIC ADIPOSE REMODELING, PRODUCING THE CLINICAL PICTURE. EACH LAYER IS INDEPENDENTLY ACTIONABLE.

PREDISPOSITION

Genetic & connective tissue.

An autosomal-dominant, sex-limited susceptibility[6] sits on a background of connective-tissue laxity — the same biology that links lipedema to joint hypermobility.[10,11] This is fixed, but it explains the family pattern and the hypermobility, and it sets the priority on protecting your daughters' early window.

TRIGGER

Estrogen & hormonal events.

The predisposition is unmasked at puberty and driven by subsequent hormonal events — pregnancy, exogenous hormones, menopause.[4,5] Your timeline maps onto this exactly: puberty onset, acceleration across two pregnancies, an 11-year drospirenone overlap. This is the modifiable layer the hormonal audit targets.

TISSUE REMODELING

Inflamed, fibrotic fat.

Lipedema tissue shows adipocyte hypertrophy independent of obesity, increased CD68+ macrophages with an M2/CD163 predominance, dilated and more permeable blood and lymphatic microvessels, and angiogenesis.[17] This remodeled tissue is what causes the pain and the diet-resistance — and what only surgery can physically remove.

What the model predicts for this case.

Three predictions follow, and each maps to a recommendation. **First,** because the diseased fat is a remodeled tissue rather than ordinary energy storage, it will not respond to diet or exercise[8,9] — so weight-focused advice (which dominated your first 14 years) is the wrong target, and conservative therapy aims at symptoms and progression, not

volume. **Second,** because estrogen drives expression,[4,5] the hormonal audit is a legitimate lever and the perimenopausal transition ahead is a period to monitor closely. **Third,** because the tissue changes are physical and largely irreversible without removal, lymph-sparing liposuction is the only intervention that addresses the established disease burden directly[1,2,3] — which is why it anchors the plan, gated behind a conservative trial. The model is not academic; it is the logic of the whole report.

SECTION 12 / INTERVENTION PRIORITIZATION

Your plan, in priority order.

Below is everything we recommend, grouped by how central it is to your plan and ordered so the steps that unlock other steps come first. The non-negotiable sequence runs across the top; the bands beneath group every intervention by role and give the timing and dependency for each.

STEP 1
Structured conservative therapy
≥ 3 months
›
STEP 2
Hormonal audit (parallel)
90-day gate
›
STEP 3
Lymph-sparing liposuction
›
ONGOING
Maintenance & surveillance

THE CRITICAL PATH — THESE RUN IN THIS ORDER. CONSERVATIVE THERAPY BOTH RELIEVES SYMPTOMS AND QUALIFIES SURGERY; THE DAUGHTERS' SCREENING RUNS ALONGSIDE, INDEPENDENT OF THIS PATH.

Foundational — start now; the rest of the plan depends on these.

WHAT IT IS	WHEN TO START	WHAT IT DEPENDS ON
Structured conservative therapy (CDT)	Week 1	Nothing — start immediately. MLD + compression + exercise + skin care.[4] Relieves symptoms now and is the documented insurer precondition for surgery.[15]
Compression garment fitting (CCL by stage)	Week 1–2	Fitted by a certified lymphedema therapist; class selected to tolerance.[4]
Vitamin D repletion + iron review	Week 1	Inexpensive, low-risk corrections; useful before surgery.
Daughters' baseline + monitoring passport	Month 1	Independent of the surgical path; opens the early-recognition window (Section 13).

High priority — the major moves.

WHAT IT IS	WHEN TO START	WHAT IT DEPENDS ON
Hormonal-audit transition trial	Month 1	Coordinated with OB/GYN re: the endometriosis indication; 90-day decision gate on symptoms and limb volume.[4,5]
Lymph-sparing liposuction (tumescent or WAL)	Month 4–9	After a documented conservative-therapy trial and with weight stability; lipedema-experienced surgeon.[1,2,3,14]

Supporting — valuable, and sequenced later or behind a dependency.

WHAT IT IS	WHEN TO START	WHAT IT DEPENDS ON

Insulin-resistance management	Month 1–2	Independent metabolic target; coordinate with primary care/endocrinology.
Formal Beighton / hypermobility assessment	Month 2	Informs PT approach and surgical positioning; flagged by the comorbidity signal.[10,11]
Anti-inflammatory dietary pattern	Month 1	For symptom and metabolic benefit — not framed as volume reduction, which lipedema fat resists.[8]
Perimenopausal monitoring plan	Ongoing	The next hormonal transition is a known risk window;[4] build the watch now.

CRITICAL SEQUENCING NOTES

Two constraints set the order. (1) The conservative-therapy trial must precede surgery — it is clinically indicated, relieves symptoms in its own right, and is a near-universal insurer documentation requirement;[4,15] skipping it risks both a worse outcome and a denied claim. (2) Surgery should be done with weight stability, since significant weight gain can drive regrowth and erode the result.[13] The hormonal audit runs in parallel on its own 90-day gate, and the daughters' screening is fully independent — it should not wait on anything else in this plan.

SECTION 13 / SPECIALIST PATHWAY

Five consults, sequenced — and what each one produces.

The pathway is built so each visit produces an input the next one needs. The named clinicians below are real, publicly recognized lipedema specialists, listed to show the caliber and type of provider appropriate to this case; in a live engagement we confirm current availability, insurance fit, and geography, and present options rather than a single name.

ROLE	REPRESENTATIVE PROVIDER	TIMING	WHAT IT PRODUCES
Certified lymphedema therapist (CLT)	A lipedema-experienced CLT (e.g., the Klose- or Vodder-trained network)	Week 1–2	Starts conservative therapy, fits compression, builds the documented trial the surgeon and insurer require.[4,15]
Lipedema-experienced physician	Karen L. Herbst, MD, PhD — adipose disorders (telehealth)[4]	Week 3–5	Confirms staging/typing, frames the medical plan, and is the diagnostic anchor for everything downstream.
Phlebology / vascular	Thomas C. Wright, MD or Lindy McHutchison, MD (vein & lymphatic)[4]	Week 4–6	Confirms the venous screen, manages any reflux, co-manages the lymphatic picture pre-surgery.
Lipedema surgery	David Amron, MD (Roxbury Institute) · Jaime Schwartz, MD · Ethan Larson, MD[4]	Month 3–4 (consult)	Assesses surgical candidacy, technique (tumescent vs WAL), staging of operative sessions, recovery plan.
Gynecology / endocrinology	Treating OB/GYN + endocrinology for the hormonal audit and IR	Week 4–6	Runs the drospirenone transition trial against the endometriosis indication; manages insulin resistance.

Why this sequence.

The CLT comes first because the conservative-therapy trial is both immediately useful and the gate to surgery — starting it on day one means the clock on the insurer's documentation requirement is already running while you get symptom relief.[15] The lipedema physician anchors the medical framing next, because the staging and typing confirmation shapes every downstream conversation. Phlebology confirms the venous screen before any operative planning. The surgical consult follows once conservative therapy is underway and the vascular picture is clear — never as a first step, because the literature and the insurers both require the conservative trial first.[4,15] Gynecology and endocrinology run in parallel on the hormonal and metabolic threads, which do not depend on the surgical timeline. Provider logistics, telehealth eligibility, and referral routing for each are in the appendix packet.

The daughters' pathway — separate and parallel.

Independent of everything above: a baseline visit with a lipedema-aware pediatric or family clinician for both daughters, and a one-page written "monitoring passport" they carry forward — recording the family history, the signs to watch for at puberty (disproportionate lower-body development, easy bruising, tenderness), and the threshold for re-evaluation. This is the entire intervention the genetics permits (Section 09), and it is the highest-leverage, lowest-cost item in the report.

SECTION 14 / MEDICAL THERAPY & SUPPLEMENT PROTOCOL

Conservative therapy, the hormonal audit, and an honest supplement stack.

Lipedema has no disease-specific drug. The therapeutic core is conservative therapy plus, for the right candidate, surgery; the medical "protocol" here is therefore a structured conservative regimen, a carefully monitored hormonal trial, and a small, honestly-scoped set of adjuncts.

FIRST LINE — CONSERVATIVE THERAPY (START WEEK 1)

Complete decongestive therapy (CDT). The standard conservative approach combines manual lymphatic drainage, compression, exercise, and skin care.[4] For lipedema specifically the goals are symptom relief and slowing progression — not volume loss, which the diseased fat resists. A randomized trial in severe lipedema found CDT and intermittent pneumatic compression each combined with exercise improved outcomes,[19] and decongestive physiotherapy has been shown to reduce the capillary fragility that drives easy bruising.[20] Protocol: CLT-directed MLD with home self-MLD; daily flat-knit compression fitted to tolerance; low-impact aquatic or cycling exercise; meticulous skin care. Reassess symptoms and limb measurements at 6 and 12 weeks.

Compression. Flat-knit garments at the compression class the CLT selects to tolerance, worn daily, with refitting as limb measurements change.[4] Compression is also the backbone of the pre-surgical "prehab" the surgeon will expect.

HORMONAL AUDIT — MONITORED TRIAL (MONTH 1, 90-DAY GATE)

Drospirenone transition. Given the tight overlap between your 11-year exposure and your steepest progression, and estrogen's established role in lipedema expression,[4,5] a supervised transition to a non-progestin option is reasonable — framed explicitly as a *trial*, because the contraceptive-specific evidence is suggestive, not conclusive. This must be coordinated with the OB/GYN managing your endometriosis, since that indication cannot simply be dropped. Decision gate: structured symptom and limb-volume tracking, formal review at 90 days, revert if no benefit or if endometriosis control suffers.

SURGERY — THE DEFINITIVE OPTION FOR ESTABLISHED DISEASE (MONTH 4–9)

Lymph-sparing liposuction. Tumescent or water-jet-assisted technique by a lipedema-experienced surgeon is the only intervention that removes the remodeled tissue; benefit is durable, with cohorts showing sustained symptom relief and reduced conservative-therapy need to 8 and 12 years.[1,2,3] Often staged across more than one session by region. Requires the documented conservative trial first and weight stability to protect the result.[13,14]

SUPPLEMENT & ADJUNCT STACK — HONESTLY SCOPED

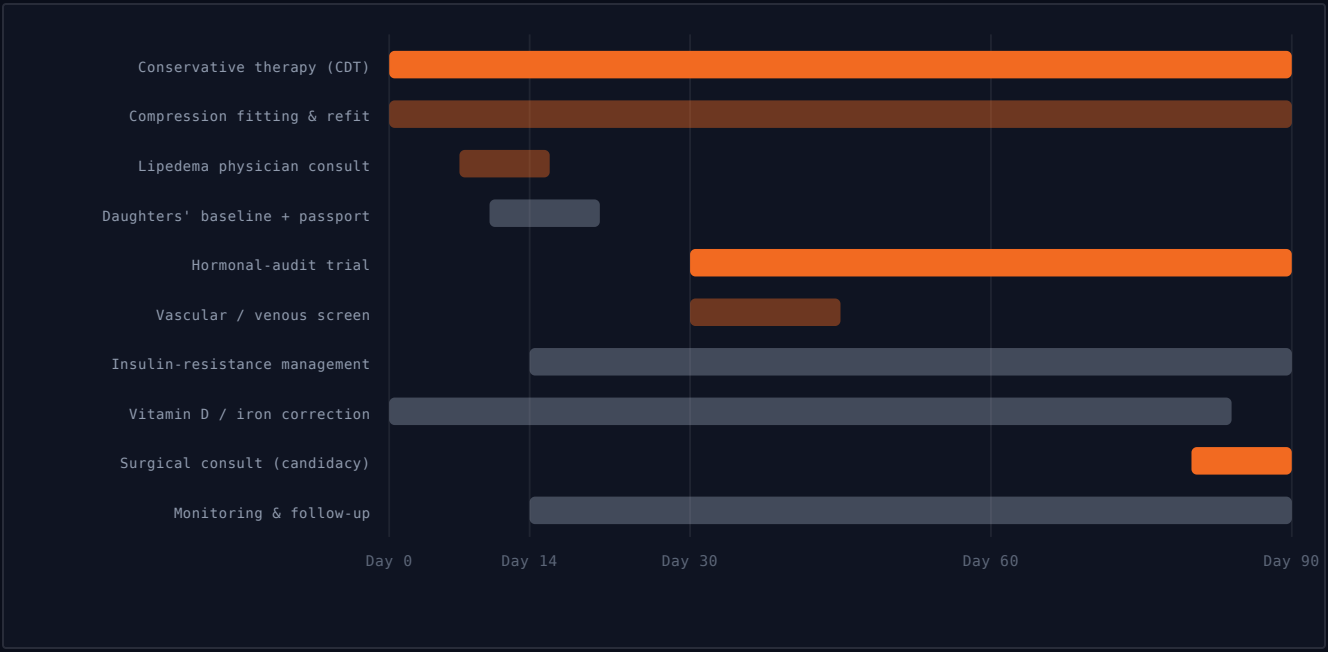
ITEM	DOSE	RATIONALE & REVIEW
Vitamin D3	2,000–4,000 IU/day	Baseline 26 ng/mL — correct the insufficiency; recheck at 12 weeks.
Omega-3 (EPA/DHA)	2 g/day	Anti-inflammatory adjunct; reasonable evidence in chronic pain, modest here.
Magnesium glycinate	300–400 mg nightly	Sleep and muscle comfort; safe long-term.
Iron (if indicated)	per ferritin recheck	Low-normal ferritin; treat only if confirmed low on repeat.

> **HONEST FRAMING**
>
> No supplement treats lipedema, and we won't imply one does. The items above address measured deficiencies and provide modest anti-inflammatory support — nothing more. The interventions that will actually change your trajectory are conservative therapy (now), the hormonal audit (a monitored trial), and surgery (the definitive step for the established tissue). Anything sold as a supplement that "reverses lipedema" is not supported by evidence, and we would rather say so plainly than pad this section.

SECTION 15 / 90-DAY EXECUTION ROADMAP

Your first 90 days, sequenced and visualized.

The integrated roadmap across the first 90 days. Where Section 12 set priority, this sets the calendar — and the conservative-therapy clock that the surgical decision depends on starts on day one. Dependencies and decision gates are reflected in the sequencing.



90-DAY ROADMAP — ACCENT BARS ARE THE CORE PATH; MUTED BARS ARE SUPPORTING WORK. SEQUENCING REFLECTS DEPENDENCIES AND THE CONSERVATIVE-THERAPY DOCUMENTATION WINDOW.

Milestone summary.

DAY	MILESTONE	WHAT CHANGES
Day 7	CDT initiated; compression fitted	Conservative-therapy documentation clock starts; symptom relief begins.
Day 14	Lipedema physician consult complete	Staging/typing confirmed; medical plan framed.
Day 21	Daughters' baseline + passport done	Early-recognition window opened; independent of surgical path.
Day 30	Hormonal-audit trial begun	Drospirenone transition started with OB/GYN; 90-day clock running.
Day 45	Vascular screen + IR plan confirmed	Venous picture clear for surgery; metabolic target underway.
Day 60	Conservative therapy mid-point review	Limb measurements reassessed; compression refit if indicated.
Day 90	Hormonal-audit decision; surgical consult	Transition continue/revert decided; surgeon assesses candidacy with conservative trial documented.

SECTION 16 / MONITORING CADENCE & PHYSICIAN QUESTION SETS

What to track, at what intervals, and what to ask.

A monitoring cadence with explicit review thresholds, and a set of pre-written questions for each specialist — so each visit is driven by your priorities, not the clock. The projection below is illustrative of the expected response if the staged plan lands as designed.

SYMPTOM TRAJECTORY — SOLID LINE IS PRE-ENGAGEMENT; DASHED LINE IS THE PROJECTED RESPONSE AS CONSERVATIVE THERAPY, THE HORMONAL AUDIT, AND SURGERY TAKE EFFECT IN SEQUENCE. ILLUSTRATIVE, NOT A GUARANTEE.

Monitoring cadence.

ITEM	FREQUENCY	THRESHOLD FOR REVIEW
Pain & symptom log	Daily → weekly	Sustained worsening over a 2-week window triggers an early review.
Limb measurements / DEXA	Limb: monthly · DEXA: 6–12 mo	Objective yardstick for conservative therapy and surgery.
Compression garment refit	Every 4–6 months	Earlier if limb measurements change significantly.
Hormonal-audit review	90-day decision point	Symptom and limb-volume change; endometriosis control.
Metabolic labs (HbA1c, insulin, lipids)	Every 3 months	Insulin-resistance management target.
Thyroid (TSH)	Every 6–12 months	Maintain euthyroid state on levothyroxine.
Daughters — pubertal watch	Annual near puberty	Any disproportionate lower-body change, bruising, or tenderness.

Pre-written questions for each specialist.

For the lipedema physician (Dr. Herbst or equivalent): "Given my Stage II, Type III presentation without secondary lymphedema, how do you weigh operating now versus continuing conservative therapy longer?" · "What specific conservative-therapy documentation will my surgeon and insurer want to see?" · "How should my hypermobility change the plan, if at all?"

For the surgeon (Dr. Amron / Dr. Schwartz / Dr. Larson): "Do you recommend tumescent or water-jet-assisted technique for my distribution, and why?" · "How many sessions do you anticipate, and how do you stage them by region?" · "What lymph-sparing steps do you take, and what is your experience with Stage II patients specifically?" · "What weight stability do you require before and after surgery to protect the result?"

For OB/GYN + endocrinology: "Can we transition off drospirenone without losing endometriosis control, and what are the alternatives?" · "How should we monitor whether the hormonal change affects my lipedema over the next 90 days?" · "Given my insulin resistance, what is your threshold for pharmacologic management, and does it interact with any surgical plan?"

SECTION 17 / APPENDIX

References, methodology, and signatures.

Every numbered claim in this report traces to the sources below. All are real, peer-reviewed publications or consensus guidelines in the lipedema literature.

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Methodology notes.

Every signal, intervention priority, and pathway recommendation in this report is grounded in the Ternary Lipedema Signal Library, which catalogs the patterns that matter for the condition across the signal categories relevant to it. Signal relevance is determined by the published evidence base combined with the case-specific personalization layer. The same workflow, the same analytic framework, and the same nine stages apply to every Precision Deep Dive, whatever the condition; what varies is the content of each section. Recommendations are graded by the strength of their source — consensus guideline, cohort study, or expert opinion — and the report distinguishes throughout between what is established, what is suggestive, and what is unknown.

> A NOTE FROM YOUR RESEARCH DIRECTORS
>
> Margaret Holloway is a composite, but the analysis applied to her case is exactly the analysis we apply to every Ternary Health Precision Deep Dive — including the discipline of saying plainly where the science runs out, as it does for genetic testing in lipedema. If you're considering engaging us, the next step is a 30-minute fit call. We'll listen to your case, tell you honestly whether we think we can help, and either propose an engagement or refer you somewhere better.

Beau Giannini, PhD · Pavel Paramonov, PhD

RESEARCH DIRECTORS, TERNARY HEALTH