PREPARED FOR MARGARET R. HOLLOWAY

A personalized action plan.

For Lipedema, Stage II, Type III (hip–thigh–calf distribution).

ENGAGEMENT
Precision Deep Dive — $6,500

CLIENT ID
TH-2026-078

DELIVERY
March 17, 2026

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COMPOSITE SAMPLE · NOT A REAL PATIENT RECORD

This report illustrates the structure and depth of a Ternary Health Precision Deep Dive. The patients and findings shown are drawn from research-validated profiles across four conditions and do not represent any single individual. Your engagement will be tailored to your specific condition, history, and goals.

SECTION 02 / TABLE OF CONTENTS

The Ternary 17 — universal section format.

Every Ternary report follows the same 17-section structure. The content and depth of each section flex with the condition and your inputs; the architecture does not. This composite features four patient profiles distributed across the sections — each labeled at the section header so you always know which condition and client is being walked through.

FEATURED CASE **MARGARET R. HOLLOWAY** · LIPEDEMA · ENGAGEMENT TH-2026-078

SECTION 03 / EXECUTIVE SUMMARY

Three findings define this case.

Ms. Holloway is a 41-year-old premenopausal woman with a 14-year diagnostic delay, newly confirmed Stage II Type III lipedema, and a hereditary pattern spanning three generations. The case turns on three decisions — sequenced, interlocking, and time-sensitive.

01 **Surgical decision: liposuction technique and timing.**

The literature distinguishes water-jet-assisted (WAL) and tumescent liposuction from cosmetic liposuction in lipedema outcomes. Choice of surgeon (Amron, Schwartz, Wright, Herbst-network providers) and technique determines five-year recurrence risk far more than timing. **Recommendation:** proceed with WAL via a lipedema-experienced surgeon within 12–18 months, using Stage II progression markers as the decision gate.

02 **Hormonal exposure audit — drospirenone continuation.**

Your 11-year drospirenone exposure overlaps the steep progression phase. The lipedema–hormonal-contraceptive literature is suggestive (not conclusive), but the burden of proof in this case favors transition to a non-progestin option. **Recommendation:** transition trial with structured symptom and limb-volume monitoring, decision point at 90 days.

03 **Daughters: early screening, early intervention window.**

With a strong hereditary pattern (mother, maternal aunt, maternal grandmother — all affected), your daughters carry meaningfully elevated risk. The window for compression, lymphatic protection, and pubertal-phase monitoring is narrow. **Recommendation:** pediatric-experienced lipedema evaluation in the next 18 months for both daughters; protocol in Section 13.

WHAT YOU RECEIVE IN THIS REPORT

A written case synthesis integrating your DEXA, labs, genetic findings, and surgical considerations into a sequenced 90-day plan. Specific named providers. Named medications and doses. Decision gates and what to monitor at each. A patient passport for daughters. Pre-written questions for each consult.

FEATURED CASE **MARGARET R. HOLLOWAY** · LIPEDEMA · ENGAGEMENT TH-2026-078

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	HEIGHT / WEIGHT	5′6″ (168 cm) / 183 lb (83.0 kg)
BMI	29.5 kg/m² (see DEXA — BMI misleads in lipedema)	GEOGRAPHY	Newton, MA
ANCESTRY	Irish (maternal), English-Scottish (paternal)	OCCUPATION	Senior product manager, biotech
DIAGNOSIS	Lipedema, Stage II, Type III	DIAGNOSED	November 2025
PRIMARY CONCERN	Progression trajectory, surgical decision	SECONDARY CONCERN	Pain management; protect daughters
REFERRAL	Lipedema Foundation community	START / DELIVERY	Feb 18 → Mar 17, 2026

History of present illness, as you described it.

You first noticed disproportion at puberty, around age 13. Your maternal grandmother and aunt had similar lower-body distribution; your mother developed the same pattern after her first pregnancy at 27. Through your twenties you were told the disproportion was poor diet or insufficient exercise, despite consistent activity (running 3–4×/week) and a Mediterranean dietary pattern. Two providers offered weight-loss recommendations; one mentioned lymphedema but did not pursue it.

Progression accelerated after your first pregnancy at age 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. Pain — deep aching, with marked easy bruising — became routine by your late thirties. Compression initiated two years ago on self-research; diagnosis confirmed at the Lipedema Foundation referral center in November 2025.

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 autoimmune dx, the hypermobility, my insulin resistance — not in isolation."*

FEATURED CASE **HANNAH M. REINHARDT** · MAST CELL ACTIVATION SYNDROME · ENGAGEMENT TH-2026-073

SECTION 05 / THE TERNARY METHOD

Evidence × Personalization × Action.

Every option in this report is weighed on three independent axes. Below, the method applied to Ms. Reinhardt's case.

EVIDENCE	PERSONALIZATION	ACTION
What the literature supports.	**What about you specifically.**	**What we recommend doing.**
For non-clonal MCAS, the Vienna consensus criteria (Valent 2019) and the proposed AAAAI workgroup criteria provide diagnostic anchors but limited treatment-specific evidence. The Ternary review integrates Akin 2017, Afrin 2020, and Brigham Mastocytosis Center guidance as the primary frame, with HαT-specific literature (Lyons 2016, Milner group) layered on top.	Your TPSAB1 copy-number variant (3 copies confirmed) places you in the HαT-positive subset, which modifies severity, anaphylaxis risk, and likely response to mast cell stabilizers. Your hEDS overlap (Beighton 7/9) and POTS overlap (head-up tilt confirmed, 2023) make this the recognized "trifecta" presentation requiring integrated management.	Three immediate moves: (1) optimize H1/H2 blockade with a specific twice-daily protocol; (2) add cromolyn before introducing any new mast cell stabilizer; (3) acquire an emergency action plan and EpiPen refresh. Sequenced over the first 30 days. Section 12 details the full intervention map; this is the entry.

Why the framework matters here.

MCAS is the condition where evidence and personalization most often diverge. The literature supports many treatments at the population level; few are consistent across individuals. Your reaction phenotype, trigger map, and TPSAB1 status shape which subset of evidence-supported interventions will actually work for you. The Ternary Method names that divergence explicitly and forces every recommendation to answer all three questions — never evidence alone.

FEATURED CASE **HANNAH M. REINHARDT** · MAST CELL ACTIVATION SYNDROME · ENGAGEMENT TH-2026-073

SECTION 06 / THE NINE-STAGE WORKFLOW, APPLIED

How your case moved through our workflow.

Every Ternary engagement runs through nine stages. Below, the version of each stage Ms. Reinhardt's case specifically produced — what we pulled, what we evaluated, what we built.

STAGE	WHAT IT DID	OUTPUT FOR THIS CASE
01 · Qualification Days 0–2	Confirm fit and complexity.	Confirmed Vienna-criteria diagnosis (Sep 2024, Brigham Mastocytosis Center). Severity history (3 ICU-level reactions in 18 months) and HαT-positive status placed the case in the high-complexity tier. Proposal sent within 24 hours.
02 · Intake & aggregation Days 3–7	Build the unified case file.	Records from Brigham, Northwestern, and primary care. Captured 4 tryptase measurements over 18 months, 2 successful 24-hour urine mediator captures, TPSAB1 testing, and complete medication trial history (16 agents across 9 years).
03 · Case structuring Days 7–10	Code to the case schema.	Reaction episodes (n = 47 over 5 years) classified by mediator type, severity, and trigger context. Trigger map built. hEDS and POTS overlap evaluated formally — both criteria met.
04 · Signal analysis Days 10–14	Apply the Signal Library.	Ternary MCAS Signal Library applied. Signals present across clinical core, mediator labs, genetics, trigger map, comorbidity, and trajectory; the central ones identified for the plan. See Section 07 for the heatmap.
05 · Evidence retrieval Days 14–19	Structured literature review.	Akin 2017 / Afrin 2020 / Valent 2019 as primary; HαT literature (Lyons 2016, Milner 2021) and MCAS-EDS-POTS reviews (Kohn 2020, Seneviratne 2017). 142 references catalogued; 38 cited directly in the plan.
06 · Pathway mapping Days 19–24	Map the option space.	Pathway built across allergy/immunology with mast cell expertise (Brigham, Stanford, BIDMC), autonomic medicine (Vanderbilt, NIH), connective tissue (Norris Lab, MUSC), and integrative pain medicine.

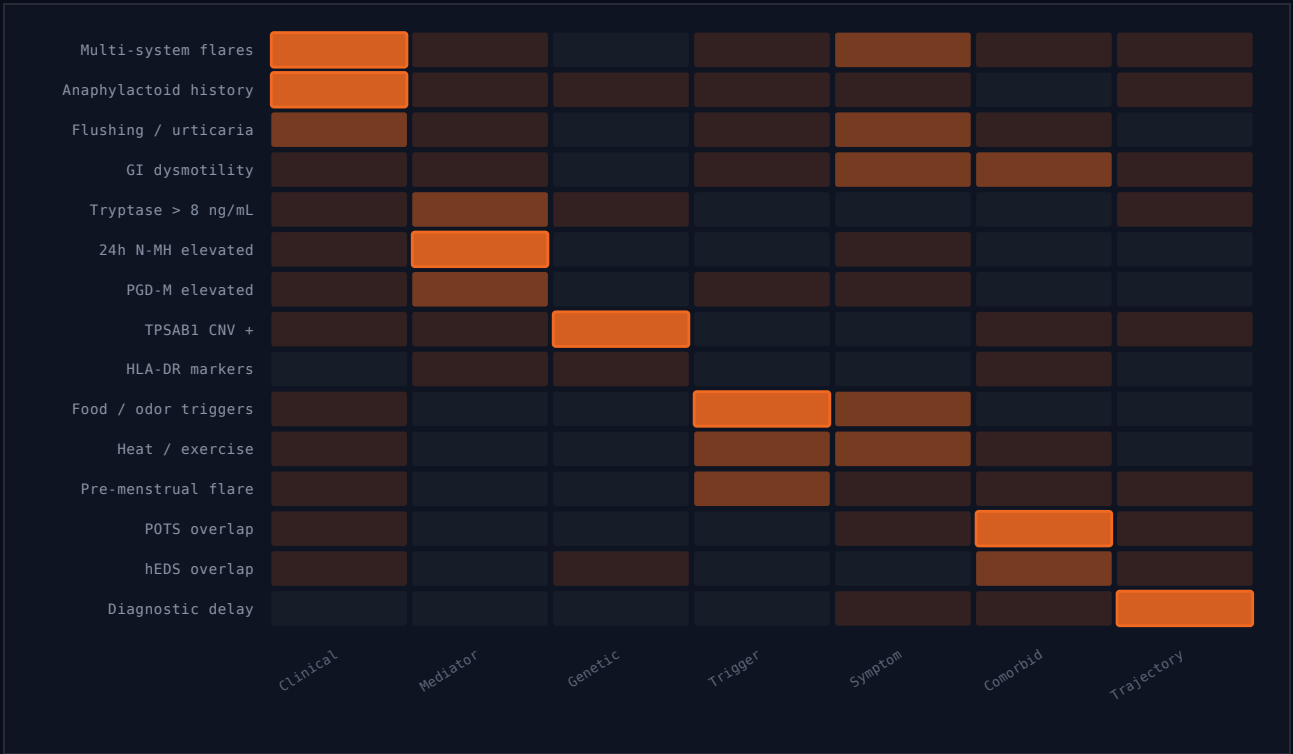
07 · Synthesis Days 24–28	Build the plan.	Interventions weighed on Evidence × Personalization × Action, then prioritized and sequenced. Dependency graph encoded — H1/H2 optimization informs cromolyn response; cromolyn response informs ketotifen or omalizumab; mediator workup informs trigger-avoidance specificity.
08 · Delivery Days 28–32	Calibrate with you.	2-hour findings call with Ms. Reinhardt, her allergist Dr. Akin (Brigham), and her PCP. Plan refined: omalizumab elevated to a 60-day decision gate given the progression pattern. Final report delivered February 26.
09 · Execution support Days 32–62	30 days of follow-up.	4 specialist consult debriefs, 2 medication titration questions, 1 reaction-episode review. Outcomes captured into the Ledger for ongoing methodology refinement.

FEATURED CASE **HANNAH M. REINHARDT** · MAST CELL ACTIVATION SYNDROME · ENGAGEMENT TH-2026-073

SECTION 07 / SIGNAL ANALYSIS & HEATMAP

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

The Ternary MCAS Signal Library codifies the patterns that matter for this condition, across the categories relevant to it: clinical core, mediator labs, genetics, trigger map, symptom burden, comorbidity, and trajectory. The heatmap shows which signals are present in your case and which are central to your plan — outlined cells are the ones your plan is built around.



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

PRESENT NOTABLE CENTRAL CENTRAL TO YOUR CASE

FEATURED CASE **HANNAH M. REINHARDT** · MCAS · ENGAGEMENT TH-2026-073

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
TPSAB1 copy-number variant positive (3 copies) CENTRAL	Confirms the HαT subset; modifies anaphylaxis risk and stabilizer response.
Multi-system flare pattern (4 systems) CENTRAL	Vienna criterion #1 met definitively; rules out single-organ alternatives.
hEDS–POTS–MCAS trifecta clustering CENTRAL	Changes management strategy; integrated workup required.
Baseline tryptase 12.4 ng/mL (sustained)	Above the 11.4 threshold; aligns with HαT phenotype and clinical severity.
Documented mediator elevation during flare (24h urine N-MH)	Objective biomarker; supports diagnosis and trigger-response evaluation.

HOW TO READ THIS

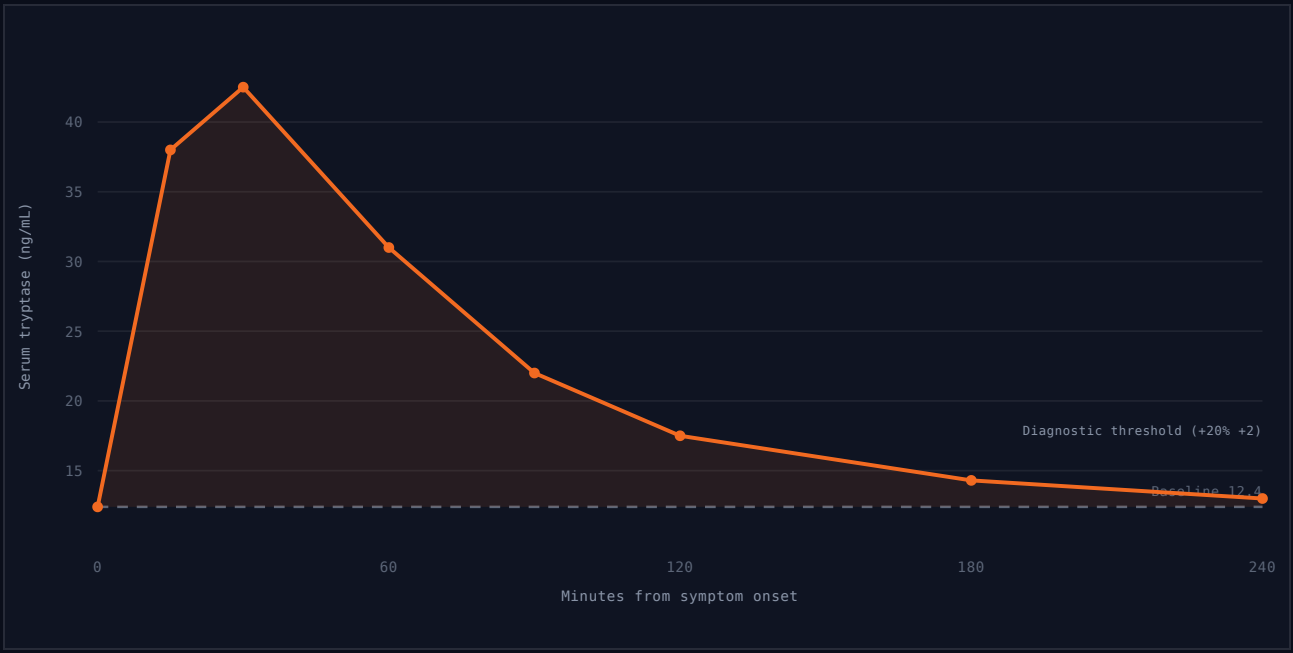
We don't assign signals a number. The Signal Library tells us which patterns matter for MCAS; reading your records tells us which are present and which are central to *you*. The heatmap is a map of that judgment, not a calculation.

FEATURED CASE **HANNAH M. REINHARDT** · MCAS · ENGAGEMENT TH-2026-073

SECTION 08 / LABORATORY FINDINGS

Mediator kinetics and baseline panels.

Mediator panels and standard chemistry across 18 months. Baseline tryptase sustained above the HαT threshold; the acute flare capture in October 2025 documented a 3.4× rise, meeting the 20% + 2 ng/mL diagnostic criterion.



TRYPTASE KINETICS — ACUTE FLARE CAPTURE, OCTOBER 2025. PEAK AT 30 MIN POST-ONSET.

Mediator panel — last 18 months.

MARKER	RESULT	REFERENCE	STATUS
Tryptase, baseline (×4)	12.4, 11.8, 12.6, 12.4 ng/mL	< 11.4 ng/mL	HIGH (SUSTAINED)
Tryptase, acute flare peak (Oct 2025)	42.5 ng/mL at 30 min	+20% + 2 above baseline	DIAGNOSTIC
24h urine N-methylhistamine	385 µg/24h	< 200 µg/24h	ELEVATED
24h urine PGD-M	1,840 ng/24h	< 1,000 ng/24h	ELEVATED
24h urine LTE4	95 pg/mg Cr	< 104 pg/mg Cr	NORMAL

Vitamin D, 25-OH	22 ng/mL	30–100 ng/mL	INSUFFICIENT
Ferritin	28 ng/mL	30–200 ng/mL	LOW-NORMAL
Total IgE	218 IU/mL	< 100 IU/mL	ELEVATED

CLINICAL INTERPRETATION

The mediator panel definitively confirms MCAS (Vienna criterion #2 met). The baseline tryptase pattern is consistent with HαT (confirmed by TPSAB1 CNV — Section 09). Vitamin D and ferritin deficiency are almost certainly contributing to mast cell instability and should be corrected before the next major intervention.

FEATURED CASE **BRUNO T. MARCHETTI** · MADELUNG'S DISEASE · ENGAGEMENT TH-2026-019

SECTION 09 / GENETIC & HEREDITARY FINDINGS

Mitochondrial inheritance pattern and family pedigree.

Madelung's Disease (MSL — multiple symmetric lipomatosis) is associated with mitochondrial DNA mutations, particularly in the alcohol-associated subtype. Mr. Marchetti carries a heteroplasmic MT-TK m.8344A>G variant (Quest Diagnostics, July 2025) — present at ~18% heteroplasmy in blood and 31% in lipoma tissue from his diagnostic biopsy.

TEST	RESULT	SIGNIFICANCE
MT-TK m.8344A>G	Heteroplasmic, 18% blood / 31% tissue	Confirmed — associated with MERRF spectrum; reported in MSL kindreds.
MT-RNR1 (full sequence)	Wild-type	No additional mitochondrial pathogenic variants.
HMGA2 (12q15)	Wild-type	Lipomatous overgrowth driver excluded.
PMP22 duplication	Negative	CMT1A excluded (peripheral-neuropathy differential).
ALDH2 (rs671)	GG (wild-type)	Normal alcohol metabolism — not protective against MSL alcohol association.
ADH1B (rs1229984)	CC (wild-type)	Slower ethanol-to-acetaldehyde conversion; relevant to alcohol counseling.

Family pedigree.

Three-generation pedigree from interview with Mr. Marchetti and his sister Sofia. Maternal lineage is consistent with mitochondrial inheritance — affected females transmit, affected males do not. No reports of MSL in the paternal lineage.

RELATIVE	STATUS	NOTES
Mother (deceased, 78)	Affected — subclinical	Distinctive "round" upper-trunk appearance from age 50; never formally evaluated.

Maternal aunt (deceased, 71)	Affected — clinical	Diagnosed with MSL at Sapienza University, Rome, 1998.
Maternal grandmother	Affected — clinical	Family records describe characteristic distribution; no genetic confirmation possible.
Sister Sofia (62)	Unaffected so far	Heteroplasmy testing recommended; will pursue.
Daughter (35)	Counseling indicated	Will inherit mitochondrial heteroplasmy. Counseling referral made.

FEATURED CASE **BRUNO T. MARCHETTI** · MADELUNG'S DISEASE · ENGAGEMENT TH-2026-019

SECTION 10 / IMAGING & BODY-SYSTEM FINDINGS

Lipomatous distribution mapped; airway and vascular involvement quantified.

MRI of the neck and upper trunk (June 2025, Rhode Island Hospital), CT angiography (August 2025, Brigham), and fiberoptic laryngoscopy (October 2025, Mass Eye and Ear). The composite picture is a Type I cervicothoracic pattern with moderate airway impingement but no critical vascular compression.

LIPOMA DISTRIBUTION DENSITY — TYPE I CERVICOTHORACIC PATTERN, ANTERIOR SCHEMATIC.

STUDY	FINDING	CLINICAL IMPACT
MRI neck (T1/T2/STIR)	Non-encapsulated lipomatous infiltration, anterior and posterior cervical compartments. Greatest dimension 8.5 × 6.2 × 4.8 cm.	SURGICAL TARGET IDENTIFIED
CT angiography neck	Mild leftward deviation of common carotid (insignificant). No IJV compression. Tracheal lumen 70% of expected.	AIRWAY BORDERLINE
Fiberoptic laryngoscopy	Glottic/supraglottic structures normal. Posterior pharyngeal wall mildly compressed. No vocal-fold paralysis.	ACCEPTABLE MARGIN
Polysomnography	Not yet performed.	OSA WORKUP PENDING

SURGICAL PLANNING IMPLICATIONS

The MRI characterizes the lesion as non-encapsulated infiltrative lipomatosis (Type I MSL), confirming that liposuction alone is insufficient. Open excision via cervical approach is the appropriate first-line surgical strategy. The carotid and IJV are accessible and not compressed; the airway margin is adequate but borderline — OSA workup must precede surgery.

FEATURED CASE **BRUNO T. MARCHETTI** · MADELUNG'S DISEASE · ENGAGEMENT TH-2026-019

SECTION 11 / DISEASE MODEL

The mitochondrial–alcohol–adipose axis in your case.

Mr. Marchetti's MSL is best understood as a three-driver process: a mitochondrial substrate, an environmental amplifier, and a downstream metabolic consequence. Each driver is independently actionable.

SUBSTRATE

Mitochondrial heteroplasmy.

MT-TK m.8344A>G at 31% tissue heteroplasmy. Impairs mitochondrial translation and oxidative phosphorylation in adipose tissue; favors brown-adipose-like dysregulation in cervical fat depots (Enzi 2002, Plummer 2013).

AMPLIFIER

Chronic alcohol exposure.

Cumulative ethanol ~125 g/day for 30 years at peak. Mitochondrial toxicity is dose-dependent and amplifies heteroplasmy expression — the most strongly correlated environmental driver of MSL severity in published cohorts.

CONSEQUENCE

Adipose dysregulation.

Impaired catecholamine-stimulated lipolysis in affected depots means these tissues don't respond to weight loss, diet, or exercise. This is why MSL patients with significant weight loss often retain the characteristic distribution — cellular biology, not adherence failure.

What this means for prognosis and intervention.

Two predictions follow. First, alcohol cessation should halt progression but will not reverse existing lipomatous tissue — surgical excision is the only route to volume reduction. Second, post-surgical recurrence risk is real (25–40% in published series at five years) but is dramatically lower with confirmed cessation. The model justifies an integrated plan: **cessation first, surgery second, surveillance third.**

FEATURED CASE **BRUNO T. MARCHETTI** · MADELUNG'S DISEASE · ENGAGEMENT TH-2026-019

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.

STEP 1	›	STEP 2	›	STEP 3	›	THEN
Alcohol cessation 12 weeks		Sleep-apnea workup (PSG)		Open surgical excision		Liposuction (selected) +6 months

THE CRITICAL PATH — THESE MUST HAPPEN IN THIS ORDER. EVERYTHING ELSE IS SEQUENCED AROUND IT.

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

WHAT IT IS	WHEN TO START	WHAT IT DEPENDS ON
Alcohol cessation — structured protocol	Week 1	Nothing — start immediately. Cessation must be demonstrated for 12 weeks before surgery.
Metabolic surveillance — HbA1c, lipid, hepatic	Month 1	Baseline before any other workup.
Metabolic / dietary plan	Month 1	Runs concurrently; supports cessation.

High priority — the major moves.

WHAT IT IS	WHEN TO START	WHAT IT DEPENDS ON
Sleep-apnea evaluation (PSG)	Month 2	Pre-surgical; the airway margin is borderline and must be planned before the OR.
Surgical excision (open) of cervical mass	Month 6	After the PSG workup and after 12 weeks of demonstrated cessation.

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

WHAT IT IS	WHEN TO START	WHAT IT DEPENDS ON

Cardiac surveillance — echo + lipid	Month 3	Coordinate with the metabolic workup.
Endocrine workup — testosterone, thyroid	Month 4	After the metabolic baseline is in hand.
Liposuction — selected lesions, post-excision	Month 9–12	Minimum 6 months after open excision; operating on healing tissue compounds complications.

CRITICAL SEQUENCING NOTES

Three constraints are non-negotiable. (1) The PSG must precede surgery — the borderline airway margin requires intraoperative airway planning. (2) Cessation must be demonstrated for 12 weeks pre-surgery — abstinence is a strong predictor of recurrence reduction. (3) Liposuction must follow open excision by at least 6 months.

FEATURED CASE **PATRICIA E. CALDWELL** · DERCUM'S DISEASE · ENGAGEMENT TH-2026-061

SECTION 13 / SPECIALIST PATHWAY

Five specialist consults, sequenced over 90 days.

Ms. Caldwell's pathway runs across the Dercum's-experienced clinician network, integrative pain medicine, endocrinology, and lymphatic-trained physical therapy. The Carolinas Healthcare network provides geographic accessibility; the Dercum Society network provides condition-specific expertise.

SPECIALTY	PROVIDER	TIMING	PURPOSE
Adipose disorders	Karen Herbst, MD PhD — Total Lipedema Care, Tucson (telehealth)	Week 2–3	Diagnostic confirmation and treatment framework.
Integrative pain medicine	Eric Lange, MD — Hansson Pain Clinic affiliate (via Dercum Society)	Week 3–4	LDN initiation, pregabalin trial planning.
Endocrinology	Atrium Health Endocrine Center, Charlotte	Week 4–6	Metabolic syndrome workup, testosterone, IGF-1.
Lymphatic-trained PT	Norton Physical Therapy, Vodder-method certified	Week 6–8	MLD course initiation, compression fitting.
Surgical (selected)	Thomas Wright, MD — Lipedema Surgical Solutions, St. Louis (consult only)	Week 10–12	Surgical assessment for selected painful nodules.

Why this sequence.

Herbst first, because her diagnostic framework anchors everything downstream — confirming the Type II generalized nodular form changes the conversation with every subsequent specialist. Lange second, because LDN takes 6–8 weeks to titrate and we want him initiating before the endocrine visit reshuffles priorities. Endocrine third, because the metabolic workup must be in hand before MLD intensity is calibrated. PT fourth, because technique selection depends on the prior three findings. Surgical consult only after medical optimization — the literature strongly favors medical-first management for Dercum's, with surgery reserved for treatment-refractory painful nodules.

FEATURED CASE **PATRICIA E. CALDWELL** · DERCUM'S DISEASE · ENGAGEMENT TH-2026-061

SECTION 14 / MEDICAL THERAPY & SUPPLEMENT PROTOCOL

A staged protocol for pain and progression.

Dercum's lacks an FDA-approved first-line treatment. The plan sequences agents with the most credible evidence and the most favorable risk-benefit profile, with explicit decision gates at each step.

First line — initiate weeks 1–12.

Low-dose naltrexone (LDN). The strongest non-trial evidence base in Dercum's is for LDN at 1.5–4.5 mg nightly (microglial modulation, endorphin upregulation). Favorable side-effect profile; access via compounding pharmacy. Titration: 1.5 mg at bedtime weeks 1–2; 3.0 mg weeks 3–4; 4.5 mg week 5. Assess at weeks 8 and 12. Avoid same-day opioids (full antagonism).

Lidocaine patch 5%. Cleanest safety profile, consistent (if modest) benefit in painful nodule clusters. Two patches per 24h, 12h on / 12h off, rotating among the most painful nodules week-by-week.

Add if first line is an incomplete response (week 12+).

Pregabalin. Lange et al. showed pregabalin + MLD outperformed either alone in a small Dercum's cohort — a real but modest signal. Start 75 mg twice daily; titrate to 150 mg bid as tolerated. Watch weight gain and sedation; stop if intolerable at 4 weeks.

Specialist-initiated, treatment-refractory only.

Mexiletine (cardiology clearance required) · IV lidocaine infusions (pain-medicine setting) · interferon-α-2b (older literature, significant adverse profile) · infliximab + methotrexate (case-series only; severe inflammatory subtype).

Supplement protocol.

SUPPLEMENT	DOSE	RATIONALE
Magnesium glycinate	400 mg nightly	Muscle relaxation, sleep; safe long-term.
Vitamin D3	5,000 IU daily	Baseline 22 ng/mL — repletion needed; recheck at 12 weeks.

Omega-3 (EPA/DHA)	2 g daily	Anti-inflammatory adjunct; reasonable evidence in chronic pain.
Alpha-lipoic acid	600 mg daily	Limited evidence; good safety. Trial at 12 weeks.
Methylated B-complex	Standard	Energy and neurologic support; verify MTHFR status.

FEATURED CASE **PATRICIA E. CALDWELL** · DERCUM'S DISEASE · ENGAGEMENT TH-2026-061

SECTION 15 / 90-DAY EXECUTION ROADMAP

Your 90 days, sequenced and visualized.

The integrated roadmap across the first 90 days. Dependencies and decision gates are reflected in the sequencing — the order is not optional.

90-DAY ROADMAP — ACCENT BARS ARE THE CORE MOVES; MUTED BARS ARE SUPPORTING WORK. SEQUENCING REFLECTS DEPENDENCIES AND DECISION GATES.

Milestone summary.

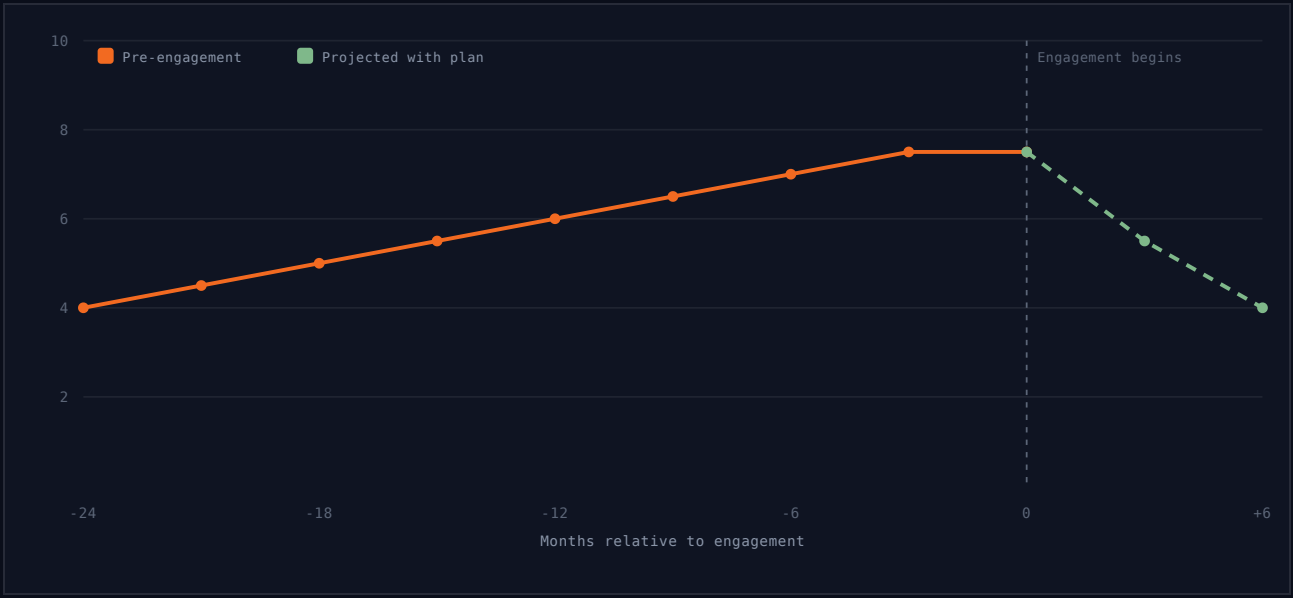
DAY	MILESTONE	WHAT CHANGES
Day 14	LDN at 1.5 mg established	Baseline pain logging begun; titration plan in motion.
Day 21	Pain medicine consult complete	LDN titration trajectory confirmed; lidocaine patch protocol finalized.
Day 28	Endocrine workup complete	Metabolic syndrome status known; testosterone, IGF-1 in hand.
Day 35	LDN at target dose (4.5 mg)	First meaningful efficacy assessment possible.
Day 60	MLD course mid-point	Lymphatic burden reassessed; compression refit if indicated.
Day 75	First-line efficacy evaluated	Decision on pregabalin add-on or continuation as is.

Day 90	Surgical consult complete	Selected-nodule surgical candidacy assessed.

FEATURED CASE **PATRICIA E. CALDWELL** · DERCUM'S DISEASE · ENGAGEMENT TH-2026-061

SECTION 16 / MONITORING CADENCE & PHYSICIAN QUESTION SETS

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



PAIN TRAJECTORY — SOLID LINE IS PRE-ENGAGEMENT; DASHED LINE IS THE PROJECTED RESPONSE IF THE STAGED PROTOCOL LANDS AS PLANNED.

Monitoring cadence.

ITEM	FREQUENCY	THRESHOLD FOR REVIEW
Pain (daily log)	Daily	Sustained high pain for 7 days triggers review.
Pain medicine follow-up	Every 6 weeks	Medication titration and side-effect review.
Endocrine labs	Every 3 months	HbA1c, lipid, hepatic, vitamin D, ferritin.
Body composition	Every 6 months	DEXA repeat to track nodule volume/distribution.
MLD course	2–3×/week first 10 weeks	Self-MLD plus monthly therapist sessions thereafter.

Pre-written questions — a sample.

For Dr. Herbst (adipose disorders): "Given my Type II generalized nodular pattern, which Dercum's subtype framework best describes my distribution and trajectory?" · "Should I be screened for the lipedema–Dercum's overlap subset given my family history?"

For Dr. Lange (pain medicine): "At what LDN dose and duration should we declare partial vs. non-response and add pregabalin?" · "My migraine history complicates pregabalin — should we trial gabapentin instead, or skip it entirely?"

For the endocrinology team: "Given the Dercum's–metabolic syndrome association, what's your threshold for initiating GLP-1 therapy, and which agent best fits my profile?"

FEATURED CASE **COMPOSITE ACROSS ALL FOUR ENGAGEMENTS**

SECTION 17 / APPENDIX

References, methodology, and signatures.

Selected references — by featured case.

Lipedema — Buck & Herbst, Plast Reconstr Surg Glob Open 2016 · Herbst et al., Phlebology 2021 · Bertsch & Erbacher, Phlebologie 2018. **MCAS —** Valent et al., J Allergy Clin Immunol 2019 · Afrin et al., Diagnosis 2020 · Lyons et al., Nat Genet 2016 · Akin, J Allergy Clin Immunol 2017. **Madelung's —** Enzi et al., Int J Obes 2002 · Plummer et al., Eur J Endocrinol 2013 · Lemaitre et al., Orphanet J Rare Dis 2021 · Schiltz et al., PRS Glob Open 2018. **Dercum's —** Hansson et al., Orphanet J Rare Dis 2012 · Lange et al., Clin Rheumatol 2013 · Beltran & Herbst, Int J Obes 2017 · Singal et al., Pain Manag 2018.

Methodology notes.

Every signal, intervention priority, and pathway recommendation in this composite is grounded in the Ternary Signal Library, which catalogs the patterns that matter for each launch condition across the signal categories relevant to that condition. Signal relevance is determined by the published evidence base (citation density, study quality, replication) combined with the case-specific personalization layer. The methodology is condition-agnostic by design — the same workflow, the same analytic framework, the same nine stages — whether the condition is rare adipose, mast cell, autonomic, connective tissue, or autoimmune. What varies is the content of each section: the signals present, the labs that matter, the imaging that's central, and the interventions that earn a place in the plan.

A NOTE FROM YOUR RESEARCH DIRECTORS

We've prepared this report with the same care we would for any client. Margaret, Hannah, Bruno, and Patricia are not real people — but the methodology applied to their composite cases is exactly the methodology applied to every Ternary Health Precision Deep Dive. 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