What Medical Evidence Can and Cannot Establish in Personal Injury Litigation

Glp-1 Agonists And Gastroparesis: Structured Causation Review
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Why this matters

GLP-1 receptor agonists *reliably* slow gastric emptying as part of their intended pharmacologic effect. The litigation question is narrower and harder: when does pharmacologic delay become a litigable injury (gastroparesis), and what evidence can credibly separate drug effect from background causes—especially diabetes-related gastric dysfunction and nonspecific GI symptoms? In practice, case viability and expert posture often turn less on whether delayed emptying is plausible (it is) and more on whether the plaintiff's record contains objective diagnostic proof and a defensible method for excluding alternative explanations.

What the evidence can reliably support

- Biological plausibility is strong. GLP-1 agonists slow gastric emptying through well-documented receptor-mediated effects on gastric motility and pyloric tone. This is central to the therapeutic mechanism, not incidental.
- Gastric slowing is class-consistent and dose-responsive (directionally). Across agents, measurable emptying delay occurs in controlled studies, with magnitude varying by dose, formulation, and patient factors.
- Baseline risk is high in the typical prescribing population. Many GLP-1 users (especially diabetics) carry independent risk of gastric motility dysfunction, creating unavoidable confounding in real-world attribution.
- Objective testing is the anchor for "gastroparesis" as a litigable diagnosis. Gastric emptying studies (typically 4-hour solid meal scintigraphy) remain the core tool for demonstrating delayed emptying in a way that can be independently evaluated.

Where claims are most vulnerable

- Diagnosis drift: side effects vs gastroparesis. Nausea, vomiting, early satiety, bloating, and abdominal pain overlap heavily with expected GLP-1 adverse effects and with other GI disorders; symptoms alone are often not etiologically specific.
- Confounding by indication and surveillance. GLP-1 users differ systematically from comparator patients (disease severity, comorbid meds, specialist exposure, testing intensity). Apparent associations can reflect detection and population selection, not drug-specific causation.
- Timing is not standardized. Symptom onset windows in reports range from days to months; "temporal proximity" is suggestive but not determinative, especially where subclinical dysfunction may predate prescribing.
- Dechallenge/recovery is ambiguous. Improvement after discontinuation can support causation, but gastroparesis symptoms fluctuate naturally and treatment changes often co-occur; lack of improvement can indicate irreversible dysfunction *or* misattribution.
- Epidemiology remains uneven. Case reports/series are inherently selection-biased; observational studies frequently rely on coding-based outcomes and imperfect exposure measurement. The denominator problem persists.

Quick screening checklist (what separates stronger vs weaker posture)

Stronger posture (more defensible)

- Objective diagnosis: documented gastric emptying study supporting delayed emptying (or equivalent objective motility test)
- Clear chronology: symptom onset and escalation mapped to initiation and/or dose escalation (not vague "months later")
- Rule-out workup: credible exclusion of mechanical obstruction and other common mimics
- Alternative causes addressed: diabetes duration/control, baseline GI history, opioid/anticholinergic exposure, other motility-affecting meds, prior GI conditions

- Coherence across records: consistent symptom narrative and testing across treating providers (not a late, litigation-driven diagnosis)
- Meaningful dechallenge evidence: documented change after discontinuation with minimal confounding treatment changes (or at least explicitly accounted for)

Weaker posture (higher challenge exposure)

- No objective testing (symptoms only; diagnosis by impression or code)
- High confounding profile: long-standing diabetes, multiple motility-impacting meds, prior GI symptoms, or inconsistent glycemic control without careful analysis
- Late documentation: long gaps between onset and evaluation; diagnosis appears after litigation context develops
- Non-specific symptom record: repeated nausea/vomiting without motility testing or coherent workup
- Attribution leaps: "GLP-1 causes gastroparesis" asserted without addressing diabetic gastropathy and other plausible etiologies

Practical implication

General causation arguments are supported by mechanistic plausibility and consistent evidence of gastric emptying delay, but specific causation is where most cases rise or fall. In the typical plaintiff profile, multiple plausible explanations exist and current diagnostic tools often cannot discriminate etiology. Opinions that claim certainty while bypassing confounding, diagnostic rigor, and objective proof are structurally vulnerable. Conversely, cases anchored in objective gastric emptying evidence and disciplined exclusion of alternatives are materially more defensible.

What the rest of the brief provides

The rest of this brief maps the domain in detail and provides:

- A structured separation of what is established vs what is contested or weak in GLP-1–gastroparesis causation

- A litigation-calibrated taxonomy of methodological vulnerabilities (diagnostic uncertainty, exposure misclassification, confounding-by-indication, protopathic bias, surveillance bias, outcome definition variance, control group problems)
- Practical, record-driven guidance for evaluating general vs specific causation posture in real cases
- A Recent Developments section covering MDL consolidation, key diagnostic proof issues, and regulatory / practice updates relevant to evidentiary posture

Download the full reference brief (PDF) if you want the complete seven-section map: the evidentiary fault lines in full, the diagnostic proof standards that drive case viability, and the method for testing causation narratives against the actual limits of the evidence base.

If you have an active matter, I also produce case-specific versions of this work. The structure stays the same; the analysis narrows to the record—exposure timeline, dose escalation, baseline diabetes and GI risk profile, testing history, competing etiologies, and the precise question you need answered. Delivered as a fixed-scope Focused Litigation Issue Brief.

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