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Chronic Liver Disease, Liver Damage and Liver Failure After Hypoabsorptive Bariatric Surgery: A Dose-Dependent Relationship and Multisystemic Consequences

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ABSTRACT

Background/Aims: Metabolic bariatric surgery remains the most potent weapon we have against severe obesity and its metabolic consequences. Yet, its effects on the liver are far from uniform. Although restrictive and mixed procedures like sleeve gastrectomy and Roux-en-Y gastric bypass consistently improve metabolic dysfunction-associated steatotic liver disease (MASLD), hypoabsorptive operations carry a distinct and troubling risk of progressive liver injury. The available evidence, drawn predominantly from case series, registry data and retrospective analyses, suggests that this risk is not an all-or-nothing phenomenon but instead follows a conceptual gradient, one that correlates with the degree of intestinal malabsorption and, more specifically, with the extent of bile acid malabsorption. This review traces the evidence for this dose-dependent relationship from the historical disaster of the jejunoileal bypass to contemporary procedures like the biliopancreatic diversion and the single-anastomosis duodenal-ileal bypass.

Materials & Methods: We explore the pathophysiological triad that drives this process—protein-energy malnutrition, bacterial overgrowth, and bile acid hepatotoxicity—supported by recent experimental evidence directly linking biliary limb length to liver injury.

Results: The review then contextualizes the discussion within associated multisystemic consequences, including de novo inflammatory bowel disease, severe metabolic bone disease, and a distinct discussion of the accelerated alcohol-associated liver disease that follows bariatric surgery.

Discussion: A synthesis of the available evidence supports the abandonment of a one-size-fits-all approach in favour of meticulous patient selection, precise and individualized surgical technique based on measured bowel length and a commitment to lifelong, intensive, multidisciplinary postoperative surveillance.

Conclusion: Hypoabsorptive bariatric procedures carry a dose-dependent risk of progressive liver injury mediated by malabsorption, bacterial overgrowth and bile acid hepatotoxicity, necessitating individualized surgical planning and lifelong follow-up.

1 | Introduction: The Paradox of Bariatric Surgery and the Liver

Bariatric surgery is established as the most effective treatment for severe obesity, producing sustained weight loss and remission of metabolic comorbidities including type 2 diabetes and hypertension [1, 2]. For patients with metabolic dysfunction-associated steatotic liver disease (MASLD), these procedures typically confer significant benefits, with regression of steatosis, inflammation, and even fibrosis documented postoperatively [3, 4]. However, this favourable outcome is not universal.

A subset of procedures, specifically hypoabsorptive operations, can paradoxically precipitate severe, progressive and potentially fatal liver injury. This duality first became evident with jejunoileal bypass, a procedure associated with unacceptably high rates of liver failure that led to its abandonment from clinical practice [5, 6]. Similar complications continue to be reported with modern operations, including biliopancreatic diversion, duodenal switch, single-anastomosis duodeno-ileal bypass and, to a lesser extent, one-anastomosis gastric bypass [7–10]. Understanding this paradox requires moving beyond the simplistic categorisation of 'malabsorptive' surgery towards a conceptual framework that recognises a spectrum of risk that appears to correlate with the degree of intestinal bypass and the resulting physiological disruption. It must be acknowledged, however, that the evidence supporting this gradient derives largely from heterogeneous case series and retrospective cohort studies, with few direct comparative trials available.

2 | The Gradient of Hepatic Risk: Evidence From Case Series, Registries and Transplant Databases

Both historical and contemporary evidence delineate a spectrum of hepatic risk associated with hypoabsorptive bariatric procedures. Table 1 summarises this gradient, comparing procedures across degree of malabsorption, reported liver risk and the strength of available evidence.

2.1 | Jejunoileal Bypass: Highest Risk

By leaving a long segment of small intestine defunctionalised, jejunoileal bypass induced profound protein-calorie malnutrition and significant bacterial overgrowth. This resulted in progressive liver failure in 5%–10% of patients, establishing this procedure as the archetypal example of the dangers of aggressive, unopposed malabsorption [5, 11].

2.2 | Biliopancreatic Diversion and Biliopancreatic Diversion With Duodenal Switch: High Risk

These procedures, while less aggressive than jejunoileal bypass, create a substantial malabsorptive state. A considerable body of literature links them to both acute and chronic liver failure, with cases frequently necessitating liver transplantation [7, 12, 13]. A Belgian survey of transplant centres identified nine patients listed for transplantation after biliopancreatic diversion, and a subsequent French nationwide study identified 50 patients with a history of bariatric surgery who required liver transplantation [14, 15]. This risk is amplified by factors including a short common channel—defined as less than 100 cm—and the presence of class IV obesity [16].

2.3 | Single-Anastomosis Duodeno-Ileal Bypass: Intermediate Risk

Single-anastomosis duodeno-ileal bypass was introduced as a modification of the duodenal switch, designed to reduce technical complexity and improve nutritional outcomes through a longer common channel, typically 250–300 cm [17, 18]. However, it is not without risk. Reports of severe protein malnutrition and liver failure after this procedure have emerged, particularly when the common channel is shorter than 250 cm or in patients with specific vulnerabilities [19, 20]. One series from a high-volume centre reported that 5.1% of patients ultimately required revisional surgery for malnutrition, and nearly a quarter of those

TABLE 1 | Spectrum of hepatic risk across bariatric procedures.

Procedure	Degree of intestinal malabsorption	Reported risk of progressive liver injury	Level of evidence
Jejunoileal bypass	Profound (90% + small bowel bypass)	Very high (5%–10% liver failure)	Historical cohort studies; procedure abandoned
Biliopancreatic diversion/ duodenal switch	Severe (common channel 50–100 cm)	High (case series of liver failure/transplant)	Case series, registry studies, transplant database analyses
Single-anastomosis duodeno-ileal bypass	Moderate–severe (common channel 250–300 cm)	Intermediate (5.1% revision for malnutrition; case reports of liver failure)	Single-centre series, case reports
One-anastomosis gastric bypass (long limb > 200 cm)	Moderate	Low-moderate (case reports with long limbs)	Case reports, systematic reviews
Roux-en-Y gastric bypass (standard proximal)	Minimal (compensated by adaptation)	Minimal (improvement typical)	Large cohort studies, RCTs
Sleeve gastrectomy	None	None (improvement typical)	Large cohort studies, RCTs

had developed some degree of liver failure [19]. These findings place single-anastomosis duodeno-ileal bypass squarely on the malabsorptive spectrum, with a real, though comparatively lower, potential for hepatic injury.

2.4 | One-Anastomosis Gastric Bypass and Long-Limb Roux-en-Y Gastric Bypass: Low-to-Moderate Risk

One-anastomosis gastric bypass, with its single loop and long biliopancreatic limb, carries a definitive risk of protein-energy malnutrition, and documented cases of subsequent liver failure exist, especially when the biliopancreatic limb exceeds 200 cm [21, 22]. Similarly, 'distal' or 'very long limb' Roux-en-Y gastric bypass, as studied in the Dutch DUCATI trial, produces superior weight loss but also increases the risk of malnutrition severe enough to require revisional surgery [23, 24]. The implication is clear: even within the Roux-en-Y gastric bypass family, increasing the malabsorptive component elevates the hepatic risk profile.

2.5 | Standard Roux-en-Y Gastric Bypass and Sleeve Gastrectomy: Minimal Risk

In stark contrast, standard proximal Roux-en-Y gastric bypass and sleeve gastrectomy carry minimal to no risk of causing de novo liver failure [25, 26]. The modest fat malabsorption observed after Roux-en-Y gastric bypass is largely compensated by intestinal adaptation, and improvements in liver histology remain the rule rather than the exception [27, 28].

This clinical spectrum provides foundational evidence for the central hypothesis: the available data suggest that the prevalence and severity of liver injury following bariatric surgery increase along a gradient commensurate with the degree of intestinal malabsorption.

3 | The Pathophysiological Triad of Liver Injury

The mechanisms driving this gradient are multifactorial and can be conceptualised as a 'three-hit' model explaining the progression from simple steatosis to liver failure. Recent experimental studies have provided causal evidence supporting each of these mechanisms.

3.1 | Hit 1: Protein-Energy Malnutrition and Lipotoxicity

Profound malabsorption of protein and calories constitutes the primary driver. Inadequate protein intake limits hepatic synthesis of very-low-density lipoprotein, which is essential for triglyceride export, resulting in substantial fat accumulation within hepatocytes [29]. This process is exacerbated by rapid peripheral lipolysis following surgery, which floods the portal circulation with free fatty acids, inducing lipotoxicity [30, 31]. The combination of substrate overload and impaired export creates conditions conducive to severe hepatic steatosis.

3.2 | Hit 2: Bacterial Overgrowth and Gut-Derived Hepatotoxins

The creation of a blind or poorly emptying intestinal limb—as in jejunoileal bypass, biliopancreatic diversion and long-limb bypasses—promotes small intestinal bacterial overgrowth [32, 33]. Gram-negative bacteria produce endotoxins (lipopolysaccharides), which, due to surgery-induced increases in intestinal permeability, translocate to the liver via the portal vein [34]. This process activates Kupffer cells through Toll-like receptor 4, triggering a pro-inflammatory cascade that drives hepatocyte injury, inflammation and fibrosis [35, 36]. The causal role of biliary limb length is powerfully illustrated by a recent experimental study, which demonstrated that extending the biliary limb led directly to small intestinal bacterial overgrowth, increased intestinal permeability, bacterial translocation to the liver and ultimately, liver failure and death [37].

3.3 | Hit 3: Bile Acid Malabsorption and Hepatotoxicity

This mechanism appears crucial and demonstrates dose-dependent characteristics. Bile acids are potent signalling molecules; however, when their enterohepatic circulation is massively disrupted—as in biliopancreatic diversion or single-anastomosis duodeno-ileal bypass with a short common channel—they are malabsorbed and spilled into the colon. Colonic bacteria convert them into hydrophobic secondary bile acids, such as deoxycholic acid, which are directly hepatotoxic [38]. Their increased return to the liver induces oxidative stress, mitochondrial dysfunction and apoptosis. Experimental data support this mechanism, showing that disruption of the enterohepatic circulation of bile acids, similar to that created by hypoabsorptive bariatric procedures, leads to the accumulation of hydrophobic bile acids in the liver, driving hepatocellular injury and fibrosis [39]. The DUCATI trial's finding that a very long Roux limb with a 100-cm common channel produced superior weight loss but also a trend towards more malnutrition-related reoperations underscores the clinical relevance of this mechanism [23].

The clinical presentation of this triad may be either acute, manifesting as what some investigators have termed 'depleted MASH' or acute liver failure within the first two postoperative years, or chronic, presenting as progressive fibrosis and cirrhosis over many years [40, 41]. Figure 1 synthesises this pathophysiological framework and its associated multisystem consequences.

4 | A Distinct Syndemic: Alcohol-Associated Liver Disease (ALD) After Bariatric Surgery

Patients with a history of bariatric surgery are at significantly higher risk of developing severe alcohol use disorder [42, 43]. This increased risk is distinct from the malabsorption-driven liver

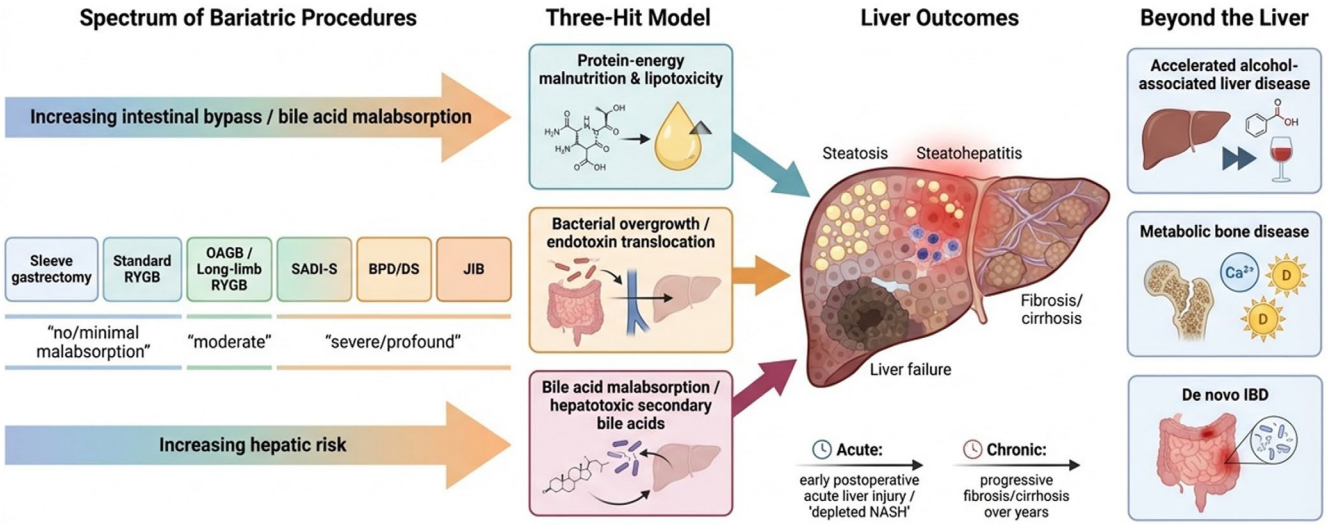

FIGURE 1 | Spectrum of bariatric procedures, three-hit model and clinical outcomes. The upper panel illustrates the gradient of hypoabsorptive procedures correlated with hepatic risk. The middle panel depicts the 'three-hit' pathophysiological model driving liver injury. The lower panel summarises hepatic and extra-hepatic consequences including progressive fibrosis, de novo IBD, accelerated alcohol-associated liver disease (driven by distinct mechanisms of altered anatomy and neurobiology) and metabolic bone disease.

injury discussed above, yet it represents an equally critical postoperative complication that can lead to accelerated liver disease.

The mechanisms driving this increased risk are twofold: altered surgical anatomy and postoperative neurobiology. Surgically, the Roux-en-Y configuration leads to accelerated gastric emptying and a higher peak blood alcohol concentration (BAC) compared to non-surgical peers, effectively converting two standard drinks into four [44, 45]. This rapid rise in BAC may enhance the rewarding effects of alcohol. Neurobiologically, animal models and human imaging studies suggest that bariatric surgery can alter central reward pathways, potentially increasing the reinforcing properties of alcohol and other substances. This shift in metabolism and reward processing creates a perfect storm, where patients both experience a heightened physiological effect from alcohol and may be predisposed to consuming more of it.

Consequently, when these patients develop ALD, the disease course is more aggressive. They present at a younger age, exhibit faster progression to cirrhosis, are more likely to present with acute-on-chronic liver failure as their first decompensating event, and demonstrate lower transplant-free survival compared to matched non-surgical patients [46, 47]. This represents a critical intersection of surgical anatomy and behavioural risk that demands routine screening and counselling. The distinction between ALD and the malnutrition-driven liver failure discussed earlier is clinically important: while both can cause end-stage liver disease, their mechanisms, risk factors and preventive strategies differ substantially.

5 | Additional Systemic Consequences of Hypoabsorption: Inflammatory Bowel Disease and Metabolic Bone Disease

The malabsorptive state that jeopardises the liver also exerts profound effects on other organ systems, creating a constellation of related complications requiring holistic management. While the following sections summarise these important associations, the reader is referred to dedicated reviews for comprehensive discussion of each topic.

5.1 | Inflammatory Bowel Disease

A growing body of evidence links bariatric surgery, particularly Roux-en-Y gastric bypass, to an increased risk of de novo inflammatory bowel disease [48–50]. A recent meta-analysis found a 17% increase in relative risk, with Crohn's disease more common than ulcerative colitis [51]. The proposed mechanisms are intimately linked to the post-surgical state: significant shifts in the gut microbiome, including increases in Proteobacteria, and alterations in bile acid signalling via FXR and TGR5, both of which play critical roles in intestinal inflammation [52, 53]. Clinically, this risk necessitates a high index of suspicion for inflammatory bowel disease in any patient presenting with persistent diarrhoea or abdominal pain after surgery, though a causal relationship requires further investigation.

5.2 | Metabolic Bone Disease

Malabsorption of calcium and vitamin D, combined with bypass of the duodenum and proximal jejunum—the primary sites of calcium absorption—leads to significant disruptions in the calcium-vitamin D-PTH axis [54, 55]. This results in high rates of secondary hyperparathyroidism and progressive bone loss, particularly at the femoral neck [56, 57]. Current guidelines advocate for routine bone mineral density monitoring and aggressive supplementation, with a low threshold for pharmacological intervention such as zoledronic acid in high-risk patients, especially postmenopausal women and men over 50 [58]. Advanced 3D-DXA studies confirm that bone loss predominantly affects

the trabecular compartment, is most severe after hypoabsorptive procedures and correlates with the degree of weight loss [59].

6 | Clinical Implications and a Call for Standardisation

Recognition of the evidence supporting this gradient of risk has profound implications for patient selection, surgical technique and long-term postoperative management.

6.1 | Patient Selection and Preoperative Counselling

Patients with pre-existing liver conditions such as advanced fibrosis, those with psychiatric vulnerabilities predisposing to alcohol use disorder or poor compliance, and individuals at high risk for metabolic bone disease should be counselled against high-malabsorption procedures [60, 61]. The decision-making process must be shared and informed by a multidisciplinary team.

6.2 | Intraoperative Decision-Making

The significant variability in total small bowel length—ranging from as little as 300 cm to over 1000 cm—mandates its routine measurement [62]. This allows for a tailored approach. If a patient scheduled for biliopancreatic diversion is found to have relatively short total bowel length, a less aggressive procedure or conversion to Roux-en-Y gastric bypass should be strongly considered to avoid an excessively short common channel [63].

6.3 | Postoperative Surveillance

The intensity and type of follow-up must be proportionate to the procedure performed.

6.3.1 | Nutritional Surveillance

Lifelong, high-dose supplementation of protein, vitamins A, D, E, K and B12 and minerals including calcium, iron, zinc and copper is mandatory [64, 65].

6.3.2 | Hepatic Monitoring

Regular monitoring of liver enzymes, albumin and international normalised ratio is crucial. A low threshold for liver biopsy in cases of unexplained liver test elevations is warranted [66].

6.3.3 | Gastrointestinal Assessment

Persistent diarrhoea should be investigated for small intestinal bacterial overgrowth, bile acid malabsorption and de novo inflammatory bowel disease [67].

6.3.4 | Metabolic and Behavioural Surveillance

Routine screening for alcohol misuse is essential. Bone densitometry should be performed at baseline and repeated at 2-year intervals, especially in high-risk groups [58].

6.4 | Management of Liver Failure

When liver failure does occur, management becomes a surgical emergency.

6.4.1 | Resuscitation

Immediate nutritional rehabilitation with total parenteral nutrition constitutes the first step, an attempt to reverse hepatocyte starvation [40].

6.4.2 | Revisional Surgery

If conservative measures fail, surgical revision is the definitive treatment. This most commonly involves lengthening the common channel, converting a single-anastomosis duodeno-ileal bypass to Roux-en-Y gastric bypass, or performing a 'kissing X' Braun enteroenterostomy [68, 69]. The evidence supporting this approach includes a series of 10 patients with post-RYGB/OAGB liver failure in whom revisional surgery to lengthen the common channel led to clinical and biochemical improvement in eight cases [70]. Case series demonstrate that these interventions effectively reverse malnutrition and liver impairment [19, 71].

6.4.3 | Liver Transplantation

In cases of irreversible liver failure, transplantation is the only life-saving option [15, 72].

7 | Conclusion

The relationship between hypoabsorptive bariatric surgery and the liver is best understood through a conceptual framework of a dose-dependent gradient of risk, supported by cumulative evidence from case series, registry data, transplant database analyses and recent experimental studies. Greater degrees of intestinal bypass are associated with higher risks of protein-energy malnutrition, bacterial overgrowth and bile acid hepatotoxicity, leading to a proportional increase in the observed prevalence of severe liver injury and failure. While acknowledging the limitations of the available evidence—predominantly derived from heterogeneous and non-comparative studies—this paradigm offers a useful lens through which to interpret postoperative complications.

This hepatic risk does not represent an isolated phenomenon but rather constitutes part of a broader syndromic cluster that includes de novo inflammatory bowel disease and severe metabolic bone disease. Additionally, the distinct entity of accelerated alcohol-associated liver disease, driven by altered anatomy

and neurobiological changes, represents another critical postoperative challenge. Collectively, these observations strongly support the abandonment of a one-size-fits-all approach to bariatric surgery in favour of meticulous patient selection, precise and individualised surgical technique based on measured bowel length, and a commitment to lifelong, intensive, multidisciplinary postoperative surveillance. Only through acknowledgment and active management of the risks suggested by this evidence can the profound metabolic benefits of hypoabsorptive surgery be realised without incurring its potentially devastating consequences.

Author Contributions

Francesco Saverio Papadia: conceptualisation, writing – original draft, writing – review and editing, supervision. **Konrad Wojcieck Karcz:** writing – review and editing, visualisation. **Andrea Pasta:** writing – review and editing, data curation. **Edoardo Giannini:** conceptualisation, writing – review and editing, supervision. All authors approved the final submitted version.

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