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# Anorexia nervosa and the liver: current understanding of pathophysiology and clinical implications

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**Abstract**

Anorexia nervosa is a severe psychiatric disorder characterized by profound caloric restriction, a distorted body image and significant weight loss. Apart from its psychological manifestations, it is associated with a spectrum of medical complications, notably involving hepatic function. Starvation-induced malnutrition promotes hepatic autophagy, leading to elevated serum aminotransferase levels, which typically correlate inversely with body mass index. These biochemical abnormalities are generally reversible with cautious nutritional rehabilitation and progressive weight restoration. Early recognition of hepatic involvement is essential to facilitate appropriate clinical monitoring and nutritional management, while excluding alternative causes of liver injury when clinically indicated. In most cases, the diagnosis is based on the clinical history, characteristic biochemical abnormalities, exclusion of other causes of liver injury, and improvement with nutritional rehabilitation. During the refeeding phase, patients are susceptible to hepatic steatosis, necessitating differentiation from starvation-related hepatitis through appropriate imaging techniques. Ongoing research is warranted to elucidate the underlying pathophysiological mechanisms of hepatic involvement in anorexia nervosa and to inform evidence-based management strategies.

**Keywords** Anorexia nervosa, hepatic manifestations, starvation hepatitis, refeeding hepatitis

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## Introduction

According to the DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, 5th edition), the main characteristics of anorexia nervosa are: 1) a limitation of energy intake compared with the body's needs, resulting in a substantially low body weight that is below what is considered minimally normal or expected for an individual's age, sex, developmental stage and overall physical health; 2) excessive preoccupation with weight and body shape, which is also characterized by a strong fear of gaining weight and an accompanying desire for a thin body; and 3) exaggerating the effect of body weight on self-esteem, without recognizing the seriousness of low body weight [1]. Anorexia nervosa affects approximately 0.05% of patients hospitalized in mental health facilities [2]. It predominantly affects females, with the highest incidence occurring between the ages of 15 and 19 years [2]. The lifetime prevalence of anorexia nervosa among women ranges from 0.9-4% [2]. Clinically, anorexia nervosa is categorized into 2 main subtypes: the restrictive type, characterized by severe caloric restriction—often between 600 and 1000 kcal per day—and the binge-eating/purging type, marked by episodes of overeating followed by self-induced vomiting or the misuse of laxatives and diuretics [1,3,4].

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The etiology is multifactorial, involving a complex interplay of genetic, neurobiological, and sociocultural factors [5-7]. Psychotherapy remains the cornerstone of anorexia nervosa treatment, with the primary goal of gradual body weight restoration [8,9]. Systematic reviews indicate that pharmacological interventions involving antipsychotics or antidepressants have not demonstrated a consistent or significant advantage over psychotherapeutic approaches in promoting or sustaining weight restoration [10]. Of particular interest are the medical complications associated with anorexia nervosa, involving those affecting the digestive system [11]. Gastrointestinal complications can be severe, including conditions such as gastric dilatation and significant liver dysfunction [11-13]. This review aims to examine the potential hepatic complications associated with anorexia nervosa, elucidate their underlying pathogenetic mechanisms, and evaluate current clinical management strategies as reported in the existing literature.

## Materials and methods

We conducted an in-depth literature search of Google Scholar, PubMed, EMBASE, the Cochrane Library, ClinicalTrials.gov and Scopus through January 2026. The search strategy included the keywords and phrases "liver," "anorexia nervosa," "liver manifestations," "hepatic manifestations," "starvation hepatitis," and "refeeding hepatitis" in order to identify relevant articles for inclusion in the final literature review.

### Body mass index (BMI) predicts liver enzyme elevation in anorexia nervosa

Previous studies have reported that 40-75% of hospitalized patients with anorexia nervosa present with elevated aspartate aminotransferase (AST) and alanine transaminase (ALT) levels [13-17]. In contrast, a retrospective study by Speranza *et al* found that only 11% and 24% of 154 outpatients had elevated AST and ALT levels, respectively [18]. This discrepancy can be partly attributed to a difference in disease severity, as outpatients exhibit a higher mean BMI (16.1±1.4 kg/m$^2$) compared to hospitalized patients, reflecting a less severe form of anorexia nervosa [13-18]. Hanachi *et al* further demonstrated that BMI is a significant risk factor for transaminasemia, showing that patients with lower BMI (11.2±2 vs. 13±2 kg/m$^2$, P<0.001) were more likely to present elevated transaminase levels [15]. AST and ALT levels tend to rise as BMI declines, and can increase up to 3 times the upper limit of normal (ULN) [13,15,17]. Gaudiani *et al* studied 25 hospitalized patients with an average BMI of 13.1 kg/m$^2$ (11-14.4) [13]. Among these, 76% (19/25) had elevated liver enzymes, and 44% (11/25) had transaminase levels elevated to 3 or more times the ULN [13]. Similarly, Gibson *et al* studied patients with severe anorexia nervosa and a median BMI of 12.1 kg/m$^2$, demonstrating elevated transaminases in 70% of participants, with 34% exhibiting levels ≥3×ULN [16]. Rosen *et al* highlighted that patients with ≥3×ULN liver enzymes had a lower BMI compared to those with only mild elevations (mean BMI 11.9 vs. 13.3 kg/m$^2$) [14]. Tomita *et al* reported that the median BMI in the group with markedly elevated ALT levels (defined as ALT>840 U/L) was 10.7 kg/m$^2$, while the median BMI in the group with moderately elevated ALT (defined as 42<ALT<840 U/L) was 13.4 kg/m$^2$ [17]. These findings reinforce the association between declining BMI and increased severity of liver enzyme elevation. Specifically, a BMI below 13 kg/m$^2$ is associated with elevated transaminase levels, which can exceed 3 times the upper normal limit, especially when BMI falls below 12 kg/m$^2$ [14,19]. Important original studies investigating the elevation of transaminase levels in patients with anorexia nervosa are summarized in Table 1.

## Hepatic complications of anorexia nervosa and pathophysiology

### Starvation-induced liver injury

One proposed pathogenetic mechanism of transaminase elevation in patients with anorexia nervosa involves liver damage from starvation-induced autophagy [19]. In the largest study to date of patients with anorexia nervosa and concomitant liver injury, liver biopsies revealed no evidence of fibrosis or inflammation; however, electron microscopy showed autophagosomes, which are formed during the process of autophagy [19]. Autophagy holds a crucial role in maintaining cellular function when deprivation of nutrients exists and provides energy by degrading cellular components [20,21]. In the case presented by Rautou *et al*, electron microscopy revealed a significant reduction in glycogen stores, along with a decrease in both the number of mitochondria and the endoplasmic reticulum [21]. While autophagy initially protects liver cells as BMI falls, it may lead to hepatic cell death if starvation continues [22]. Thus, in anorexia nervosa patients, transaminase elevation results from autophagy-induced hepatocyte death, rather than liver necrosis [23]. The elevated vitamin B12 concentrations frequently observed in patients with anorexia nervosa may reflect the release of stored vitamin B12 from the liver, and further support the predominant role of autophagy in hepatic injury among these patients [24,25]. Upon refeeding, transaminase levels tend to decrease, indicating that autophagy is reversible, unlike liver necrosis [19].

The presence of nonalcoholic fatty liver disease (NAFLD) in the starvation phase of anorexia nervosa, due to impaired degradation and secretion of lipids, does not seem to be a significant cause of elevated transaminases [26,27]. It should be mentioned that, in a study conducted by Fanin *et al*, only 1 of 16 patients with anorexia nervosa and elevated ALT exhibited evidence of NAFLD on magnetic resonance imaging during starvation [27]. Results in mouse models further reinforce the view that starvation

**Table 1** Important original studies investigating the elevation of transaminase levels in patients with anorexia nervosa

| Author [ref.] | Number of patients (female) (hospitalized patients or outpatients) | Mean BMI (kg/m$^2$) | Number of patients with elevated transaminase levels (%) | Number of patients with elevated transaminase levels ≥3 × ULN (%) |
|---|---|---|---|---|
| Gaudiani *et al* [13] | 25 (22) (hospitalized) | 13.1 | 19 (76%) | 11 (44%) |
| Rosen *et al* [14] | 181 (162) (hospitalized) | Patients with severely elevated liver enzymes (≥3×ULN) had a lower BMI (11.9 vs. 13.3) compared with the rest of the cohort | 109 (60%) | 64 (35.4%) |
| Hanachi *et al* [15] | 126 (117) (hospitalized) | Lower BMI (11.2 vs. 13, *P*<0.001) was a risk factor for hypertransaminasemia (P<0.001) | 54 (42.9%) | Not specified |
| Gibson *et al* [16] | 281 (256) (hospitalized) | 12.1 | 126 (45%) | 95 (33.8%) |
| Tomita *et al* [17] | 37 (37) (hospitalized) | 13 | 13 (35.1%) | Not specified |
| Speranza *et al* [18] | 154 (154) (outpatients) | 16.1 | 17 (11%) and 37 (24%) of outpatients had elevated AST and ALT levels, respectively | Not specified |
| Cuntz *et al* [19] | 1) 3755 (3598) (hospitalized) 2) subgroup of 142 severely malnourished females with BMI ≤13 | 15.4 (overall), 11.5 (subgroup) | More than 50% of the patients of the subgroup had elevated transaminases | Not specified |

*BMI, body mass index; ULN, upper limit of normal; AST, aspartate aminotransferase; ALT, alanine transaminase*

increases liver damage, primarily through enhanced autophagy, which is accompanied by a parallel reduction in liver fat [28,29]. Nevertheless, given that, in developing countries, early nutrient deficiencies can result in malnutrition-related hepatic steatosis in conditions such as marasmus and kwashiorkor, further insight into liver damage mechanisms in malnourished patients is considered imperative [29].

### Liver injury during refeeding

An increase in transaminases can occur during refeeding, although it is milder compared with the elevation observed in starvation-induced hepatitis [15,30,31]. Refeeding-related injury results from subsequent insulin secretion, with a parallel excessive accumulation of glucose and fat in liver cells, following a rapid increase in caloric intake, even if the calories provided are still below the normal requirement [32,33]. The aforementioned pathogenetic mechanism has also been suggested for the development of hepatic steatosis in patients undergoing parenteral nutrition [34].

### Alternative mechanisms of liver injury

Other notable potential mechanisms of liver damage in patients with anorexia nervosa are drug-induced liver injury due to the frequent use of methamphetamines, laxatives, acetaminophen, antidepressants and antipsychotics, as well as the potential coexistent severe hypoperfusion that can be detected in patients with extreme anorexia nervosa [31,35,36].

### Other liver-related biochemical abnormalities beyond aminotransferase elevation

Elevated alkaline phosphatase (ALP) and gamma-glutamyl transferase has been also described in patients with anorexia nervosa, possibly caused by the aforementioned mechanisms of liver injury [14,18]. However, hypophosphatemia, as well as the disturbed bone metabolism observed in anorexia nervosa, also contribute to the elevation of ALP [37,38].

Albumin in the majority of patients with anorexia nervosa is within normal levels, and does not correlate with weight status [39,40]. It has been observed that malnutrition without inflammation typically does not cause a significant decrease in serum albumin [41]. This is probably due to compensatory reductions in both the fractional catabolic rate of albumin and resting energy expenditure [41]. Given the limited use of the albumin value for evaluation in patients with anorexia nervosa, and the fact that many of them exhibit concurrent dehydration, Yoshida *et al* suggested that hematocrit (HCT)-adjusted serum albumin could be an alternative nutritional marker [42]. The same investigators also noted a statistically

significant correlation between HCT-adjusted serum albumin and ferritin, an iron-storage protein, suggesting that malnutrition might lead to a defect in iron transport and reactive oxygen species production, which could be an additional pathogenic mechanism of liver injury in patients with anorexia nervosa [42].

Of particular interest is the use of prealbumin as predictive marker of medical complications in anorexia nervosa [43,44]. According to Rosen *et al*, patients with severely elevated enzymes (more than 3 times the ULN value) have lower prealbumin levels at admission [14]. In addition to this, lower prealbumin levels and severe liver dysfunction are both linked to a higher risk for hypoglycemia [13,14,16,44]. These findings are consistent with a study involving 545 patients (average BMI at admission 13 kg/m$^2$) diagnosed with anorexia nervosa and/or restrictive eating disorders, which found that hypoglycemia was common until day 7, and was correlated with both elevated liver function tests and low prealbumin levels [45]. Hypoglycemia in patients with anorexia nervosa is a complication that can result in coma and death [46,47]. Pathogenetic mechanisms for the appearance of hypoglycemia in starvation-related liver damage in patients with anorexia nervosa involve impaired gluconeogenesis and depletion of glycogen stores [48,49]. Matsunaga *et al* highlighted that a confirmed reduction in liver volume can be observed through contrast tomography (CT) shortly before or after the onset of hypoglycemia, and serves as a prognostic indicator for complications and poor outcomes [50]. This reduction in liver volume may indicate that the liver was consumed as a last energy reserve to ensure daily survival [50].

Elevations of international normalized ratio (INR) and coagulopathy can be observed in anorexia nervosa [51]. Potential mechanisms of INR elevation include an extreme loss of adipose tissue and low-fat intake that may lead to poor absorption of fat-soluble vitamin K [51]. A high INR is also a prognostic factor for low thrombopoietin (TPO) and thrombocytopenia [52]. As nutritional status improves, a rapid increase in TPO is observed, accompanied by a rise in platelet count [53]. Table 2 presents the significant pathogenetic mechanisms underlying liver injury and elevated transaminases in anorexia nervosa.

**Table 2** Important pathogenetic mechanisms contributing to the elevation of transaminases in anorexia nervosa

| Mechanism | Key findings |
|---|---|
| Starvation-induced autophagy | - Breakdown of cellular structures (mitochondria, endoplasmic reticulum) for energy during starvation, leading to hepatocyte damage<br>- Presence of autophagosomes |
| Refeeding-related fatty liver infiltration | - Rapid increase in insulin→lipid accumulation→mild hepatocyte injury<br>- Impaired lipid degradation/secretion |
| Drug-induced liver injury | From methamphetamines, laxatives, acetaminophen, antidepressants, antipsychotics |
| Hypoperfusion-related injury | Severe dehydration/hypotension leading to hepatic hypoperfusion and injury |

### Rare liver manifestations of anorexia nervosa

Other rare liver manifestations of anorexia nervosa include portal hypertension, gastroesophageal varices, hyperplastic nodules of the liver and ascites [21,54-59]. Acute hepatocellular injury, hypoalbuminemia and possible chronic inflammation of the intestinal serosa, mesentery and peritoneal surface of the liver may contribute to the formation of ascitic fluid, although these mechanisms are inferred from isolated cases and do not allow conclusions regarding causality [58]. Regarding portal hypertension and gastroesophageal varices, proposed pathogenetic mechanisms include abnormal hepatic circulation related to prolonged laxative abuse, increased intestinal permeability with endotoxin influx into the portal circulation due to malnutrition, and the NAFLD-like histopathology observed in anorexia nervosa; however, these hypotheses are based on individual case reports [56,60-62]. In fact, a liver biopsy performed in a case report presented by Takata *et al*, describing gastroesophageal varices in a patient with anorexia nervosa, revealed microscopic findings similar to those associated with NAFLD [57]. Interestingly, the presence of liver hyperplastic nodules described in the same case report may have been a consequence of anomalous portal tract syndrome, suggesting a potential irregular distribution of portal blood flow in patients with anorexia nervosa [63].

## Differential diagnosis of liver injury in patients with anorexia nervosa

The differential diagnosis of liver injury in patients with anorexia nervosa remains challenging.

### Ruling out other causes of liver injury

Primarily, a thorough exclusion of alternative causes of liver injury—i.e. ischemic hepatitis (laboratory findings such as increased LDH, high elevation of transaminases >1000), viral hepatitis (viral markers for hepatitis A, B, C and E viruses), autoimmune hepatitis (antinuclear antibody, anti-smooth muscle antibody, anti-liver/kidney microsomal antibody), metabolic diseases (evaluation of ferritin, ceruloplasmin value, etc.), celiac disease (anti-transglutaminase antibody), drug-induced liver injury (medical history of drugs such as methamphetamines, laxatives, acetaminophen, antidepressants and antipsychotics), alcohol consumption, hepatic vascular and biliary disorders (CT scan, abdominal triplex ultrasound, magnetic resonance cholangiopancreatography, abdominal ultrasound)—is essential in patients with anorexia nervosa and elevated transaminases [31].

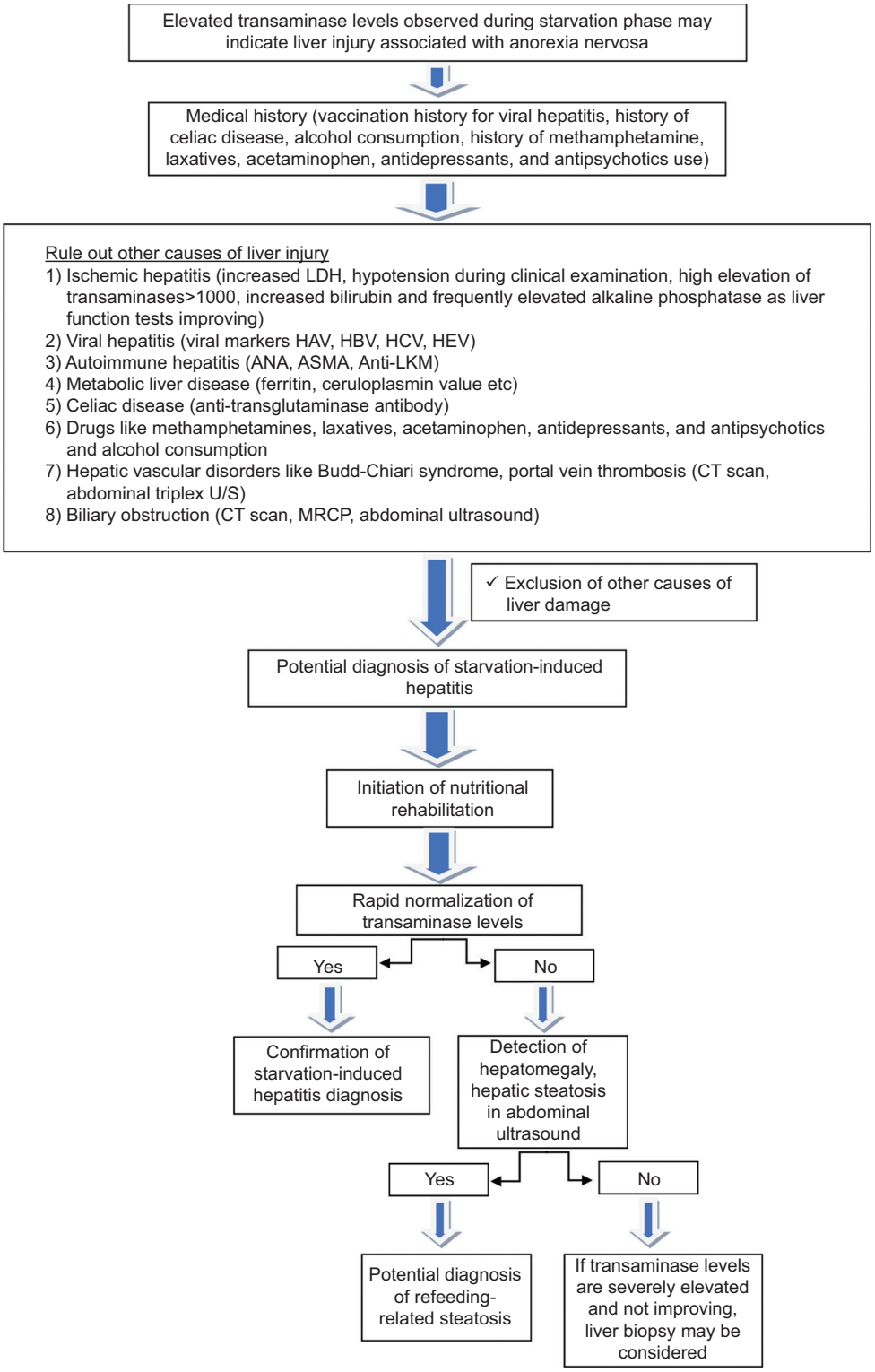



**Figure 1** Diagnostic approach to liver damage associated with anorexia nervosa during starvation phase
*ANA, antinuclear antibody; Anti-LKM, anti-liver/kidney microsomal antibody; ASMA, anti-smooth muscle antibody; CT, computed tomography; HAV, hepatitis A virus; HBV, hepatitis B virus; HCV, hepatitis C virus; HEV, hepatitis E virus; LDH, lactate dehydrogenase; MRCP, magnetic resonance cholangiopancreatography; U/S, ultrasound*

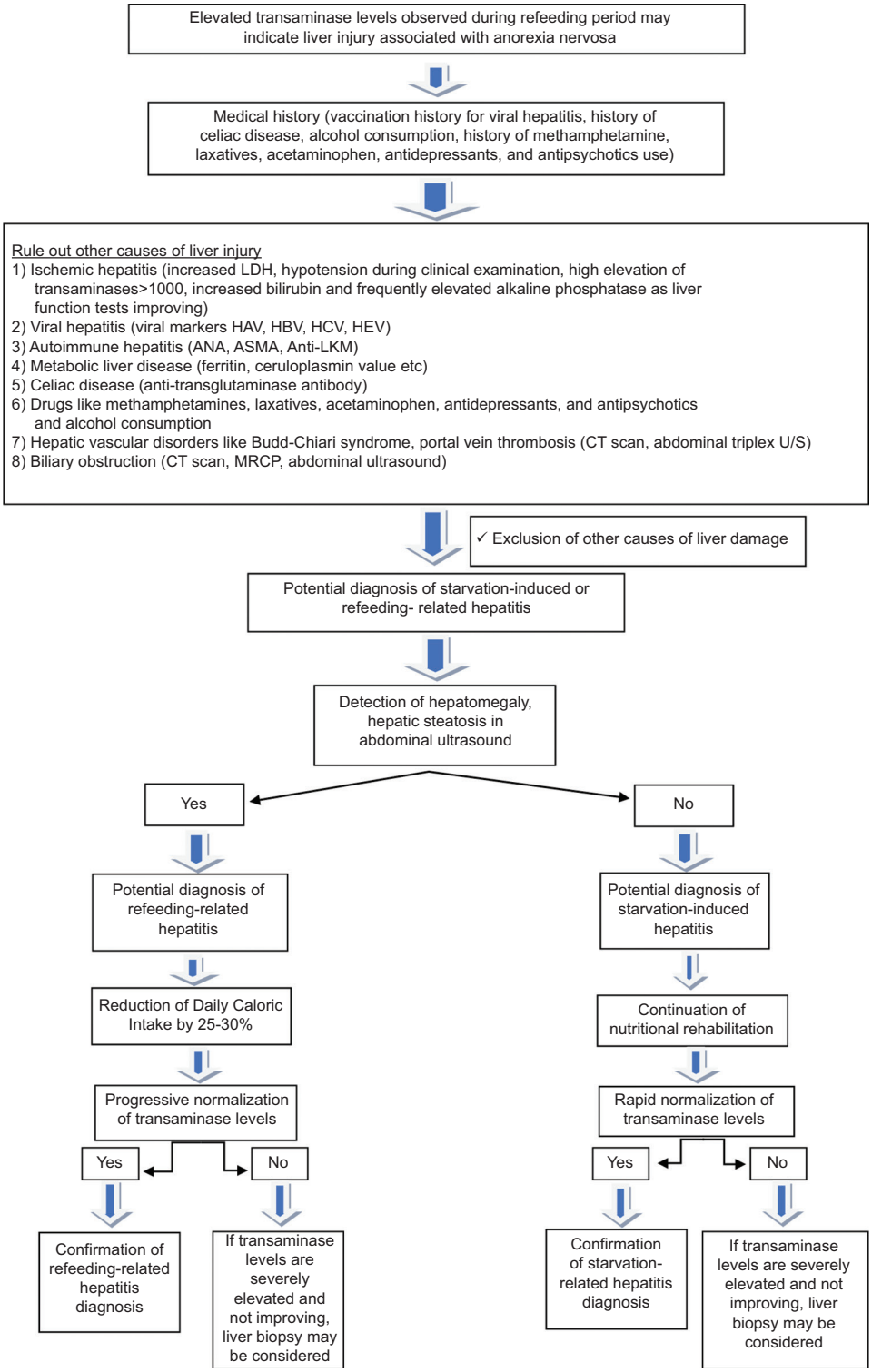



**Figure 2** Diagnostic approach to liver damage associated with anorexia nervosa during refeeding period
*ANA, antinuclear antibody; Anti-LKM, anti-liver/kidney microsomal antibody; ASMA, anti-smooth muscle antibody; CT, computed tomography; HAV, hepatitis A virus; HBV, hepatitis B virus; HCV, hepatitis C virus; HEV, hepatitis E virus; LDH, lactate dehydrogenase; MRCP, magnetic resonance cholangiopancreatography; U/S, ultrasound*

### Differentiating between starvation-induced and refeeding-related hepatitis diagnosis

After the aforementioned causes of liver injury have been ruled out, the differential diagnosis between starvation-induced and refeeding-associated hepatitis is clinically important, as the underlying pathophysiology, prognostic implications and management strategies differ significantly.

Starvation-induced hepatitis is generally considered a self-limiting and adaptive response to extreme malnutrition [31]. Elevated transaminase levels during the starvation phase raise suspicion for starvation-related hepatitis, which is typically characterized by a rapid improvement in aminotransferase levels following the initiation of refeeding [19].

Persistence of elevated transaminase levels during refeeding, or an early rise in transaminases in patients who initially had normal levels before the onset of nutritional rehabilitation, raises suspicion for refeeding-related hepatitis, and warrants careful monitoring for refeeding syndrome [51,64]. Abdominal ultrasound is essential in the differential diagnosis between starvation-induced and refeeding-related hepatitis, as the presence of hepatomegaly and hepatic steatosis during the refeeding period supports the diagnosis of refeeding-related hepatitis [14,27,33].

Delayed elevations in aminotransferase levels during the refeeding period have also been reported rarely, even in patients who initially present with normal values at admission, and probably reflect ongoing starvation-induced hepatic autophagy [60,65,66]. In such limited cases, it is possible that the initial caloric intake following the onset of refeeding was insufficient, or that hepatic recovery was delayed, resulting in liver dysfunction manifesting later in the course of nutritional therapy [65].

Liver biopsy can provide valuable insights into the degree and nature of hepatic injury in patients with anorexia nervosa [19,21]. Notably, the histological liver damage may be disproportionately severe compared to the degree of transaminitis observed in laboratory tests [67]. Histopathological findings differ notably between starvation-induced hepatitis and refeeding related hepatitis [31]. In starvation-induced hepatitis, liver biopsy typically reveals mild lobular inflammation with glycogen depletion in the cytoplasm of hepatocytes (hepatocellular ballooning), while liver biopsy in patients with refeeding-related hepatitis commonly reveals fatty infiltration with moderate portal inflammation, hepatocellular ballooning and increased glycogen deposition [19,31]. Nevertheless, liver biopsy is not recommended as a routine diagnostic procedure in patients with anorexia nervosa, given its invasive nature, associated costs and potential complications [51]. It may be considered in patients with persistently marked transaminase elevation despite adequate nutritional rehabilitation, particularly if an alternative or coexisting cause of hepatic injury is suspected [51,67]. The diagnostic approaches to liver injury associated with anorexia nervosa during the starvation and refeeding phases are illustrated in Fig. 1 and 2, respectively.

## Management strategies

A comprehensive multidisciplinary approach combining nutritional rehabilitation, medical care and psychological support is crucial for improving outcomes in patients with anorexia nervosa and associated liver dysfunction. With proper nutritional support, weight gain is achieved, leading to metabolic stabilization and a reduction in the high transaminase levels associated with starvation [19]. Natural feeding holds a primary role in the nutritional support of patients with anorexia nervosa [65]. When combined with nutritional supplements and nasogastric feeding, natural feeding is more effective, and is associated with a shorter duration of hospitalization [65].

In recent major studies, patients followed a daily caloric intake ≥2000 kcal/day [19,68]. According to Cuntz *et al*, a high-calorie diet at the start of nutritional rehabilitation is associated with rapid normalization of transaminase levels, within 4 weeks after initiation of refeeding, probably reflecting restoration of metabolic stability and reduced autophagy [19].

In rare cases of refeeding-related hepatic steatosis, a reduction of 25-30% in daily calorie intake is recommended [31]. Supplementation with thiamine, potassium, phosphate and magnesium, alongside gradual rehydration by sodium chloride (NaCl), is also necessary in patients with refeeding syndrome [31]. Fortunately, normalization of liver function occurs in nearly all patients with anorexia nervosa after discreet refeeding [19,51,69,70].

## Concluding remarks

Liver complications in anorexia nervosa primarily involve starvation-induced and refeeding-related hepatitis. Elevated aminotransferases during the starvation phase are associated with malnutrition-induced autophagy, which is reversible with the initiation of nutritional support and gradual regaining of weight. Early recognition of hepatic involvement is essential to facilitate appropriate clinical monitoring and nutritional management. It is necessary to carefully monitor refeeding for early detection of refeeding-related hepatic steatosis, which should be distinguished from starvation-associated hepatitis through imaging. Overall, a multidisciplinary approach, involving nutritional rehabilitation, medical management and psychological support, is essential to improving outcomes in patients with anorexia nervosa and associated liver dysfunction. Future studies should further explore the underlying pathophysiology of autophagy, highlight new potential biomarkers for anorexia nervosa and develop targeted therapeutic strategies [71,72].

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