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Review

Liver Transplantation in Autoimmune Liver Disease

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Abstract

Autoimmune liver diseases, including autoimmune hepatitis, primary biliary cholangitis, and primary sclerosing cholangitis, are the fourth most common cause of transplantation worldwide. Although immunosuppressive therapies can induce remission or slow disease progression, transplantation remains the definitive treatment for patients with end-stage liver disease, intractable symptoms, or acute liver failure. This chapter provides a comprehensive overview of pre- and post-transplant evaluation processes in patients with autoimmune liver diseases.

Keywords: Autoimmune liver disease, chronic rejection, liver transplantation, immunosupression

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Autoimmune liver diseases (AILDs), including autoimmune hepatitis (AIH), primary biliary cholangitis (PBC), and primary sclerosing cholangitis (PSC), are chronic conditions that can culminate in cirrhosis, hepatic decompensation, and ultimately necessitate liver transplantation (LT).[1] Although LT is frequently performed in the context of end-stage liver disease or hepatocellular carcinoma (HCC), AILDs also present unique, disease-specific indications. For instance, AIH may lead to fulminant hepatic failure requiring urgent transplantation, whereas patients with PBC or PSC may suffer from debilitating pruritus or repeated episodes of cholangitis that are refractory to medical therapy.[2] In the case of PSC, additional concerns such as biliary epithelial dysplasia or early cholangiocarcinoma (CCC) may warrant consideration for LT.[3] These diverse clinical scenarios underscore the complex decision-making involved in managing AILDs and determining transplant candidacy In this chapter a comprehensive review of pretransplant evaluation and posttransplant management will be given.

Waitlist Mortality and Transplant Evaluation

Several studies have demonstrated that patients with AILDs face higher waitlist mortality than those with other liver disease etiologies, largely due to a lower likelihood of receiving a transplant.[4, 5] Zhou et al. reported a waitlist mortality rate of 20% for individuals with PBC, which was notably higher than the rates observed for alcoholic liver disease (ALD) at 13% and metabolic dysfunction-associated fatty liver disease (MAFLD) at 18% under the MELD allocation system.[6] Similarly, Singal et al. found that the cumulative 90-day waitlist mortality for patients with PBC reached 20.1%, the highest among all etiologies examined.[7] A significant contributing factor is that most patients with AILD are women who have historically been disadvantaged by the MELD-Na scoring system. Because serum creatinine is used to estimate kidney function and tends to overestimate renal function in women, their mortality risk may be underestimated, resulting in a lower prioriti-

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zation for transplantation. Newer allocation models, such as MELD 3.0, aim to address these limitations.[8] However, further studies are needed to determine whether MELD 3.0 offers superior mortality prediction compared with MELD-Na or the original MELD system in patients with AILD.

Patients with AIH may present with a spectrum of acute liver injury, including acute severe autoimmune hepatitis (ASAIH), acute liver failure (ALF), or acute-on-chronic liver failure (ACLF).[9] ASAIH is typically characterized by jaundice, an international normalized ratio (INR) between 1.5 and 2, the absence of hepatic encephalopathy, and no evidence of chronic liver disease on biopsy. In contrast, ALF is defined by the presence of hepatic encephalopathy and an INR greater than 2. ACLF occurs in individuals with underlying chronic liver disease who develop an acute deterioration.[10]

While corticosteroid therapy is often effective in ASAIH, the outcomes in patients with ALF or ACLF are significantly worse, and these patients should be promptly referred for LT. Although most individuals with ASAIH respond well to steroids, with transplant-free survival rates ranging from 52% to 95.2%, a subset of patients fails to respond to treatment.[9]

To help identify high-risk patients, the Survival and Prognostic Factors for Acute Severe AIH (SURFASA) score was developed from a retrospective multicenter French cohort. This score, calculated as [6.80 + 1.92 × (Day 0 INR) + 1.94 × (% change in INR by Day 3) + 1.64 × (% change in bilirubin by Day 3)], can predict poor outcomes. A score above 1.75 is associated with an 85–100% risk of death or need for transplantation.[11]

Given that corticosteroid therapy increases the risk of infection, particularly if transplantation becomes necessary, the optimal steroid dosing strategy remains controversial. In the absence of definitive studies comparing 0.5 mg/kg/day versus 1 mg/kg/day, a lower dose of 0.5 mg/kg/day may be more appropriate, as it could provide therapeutic benefit while minimizing the risk of infection.[12]

Historically, requests for exception points, frequently due to severe pruritus, were assessed by regional review boards, resulting in inconsistencies across different geographic regions. The implementation of the National Liver Review Board (NLRB) in 2019 aimed to centralize and standardize these evaluations nationwide. However, current NLRB guidelines do not consider pruritus alone to be a sufficient justification for granting a MELD exception, stating that evidence is lacking to support such a policy.[13] Therefore, although severe pruritus can significantly impair quality of life and may warrant liver transplantation, it is not recognized as a formal criterion for MELD exception and must be submitted as an individual appeal.[14]

MELD exception points for liver transplantation may be awarded to PSC patients who have had two or more hospital admissions within a year for acute cholangitis, provided that there is documented bloodstream infection or sepsis requiring vasopressor support for hemodynamic instability. Patients with a confirmed diagnosis of CCA are also eligible for MELD exceptions, according to current guidelines.[15]

Risk of Rejection in Autoimmune Liver Diseases

Patients with AILD have a higher risk of rejection after liver transplantation, particularly those with AIH, who show increased rates of early and late TCMR and chronic rejection compared to non-immune liver diseases. Chronic rejection occurs in approximately 15% of AIH recipients, with a higher risk observed in younger patients. PBC and PSC are also associated with increased early and late TCMR, although chronic rejection rates are closer to those of non-immune diseases. Antibody-mediated rejection is not linked to AILD, but plasma cell-rich rejection (previously called de novo AIH) occurs in 3–5% of non-AIH transplant recipients.[16]

Post-Transplant Outcomes by Disease Subtype

Autoimmune Hepatitis

In North America, AIH has consistently accounted for approximately 4-6% of liver transplants (LT).[1] Data from the European Liver Transplant Registry (ELTR) spanning 1988 to 2016 indicate that AIH was responsible for 2% of liver transplants and 5% of transplants due to cirrhosis.[17] While liver transplantation for AIH is associated with favorable outcomes, as evidenced by the 5- and 10-year survival rates of 86% and 72%, respectively,[18] some studies have indicated a higher risk of mortality and reduced graft survival in AIH patients than in those with other chronic liver diseases.[19, 20]

Recurrent autoimmune hepatitis (rAIH) develops in approximately 20–30% of liver transplant recipients within 5 years, with rates varying depending on whether biopsies are performed only for clinical indications or routinely as part of protocol surveillance.[18, 21, 22] The diagnostic criteria for rAIH are the same as those for the primary disease and include positive autoantibodies, elevated immunoglobulin G (IgG) levels, and characteristic histological findings, such as lymphoplasmacytic portal inflammation with interface hepatitis and lobular collapse with necrosis.[23]

A key clinical challenge is to distinguish rAIH from T-cell–mediated rejection (TCMR). TCMR is characterized by a mixed inflammatory infiltrate, bile duct injury, and endothelialitis, in contrast to the more plasma cell–rich infiltrates

typical of rAIH. Protocol biopsies are particularly valuable because rAIH may be present even in patients with normal liver function tests at 1-, 5 and 10 years interval.[24]

It is also important to differentiate rAIH from de novo AIH, which occurs more than six months after transplantation in patients without a prior diagnosis of AIH. De novo AIH typically demonstrates plasma cell–rich portal inflammation on histology.[25]

Reported risk factors for rAIH include younger recipient age, higher disease activity prior to transplantation (elevated IgG, markedly raised transaminases, and moderate-to-severe inflammation in the explant), donor–recipient sex mismatch, use of mycophenolate mofetil (MMF), and withdrawal of corticosteroid therapy (Table 1).[18, 22, 26-28] A large multicenter study identified a link between MMF use and rAIH; however, some researchers suggest that this association may reflect differences in practice patterns or inter-center variables rather than a direct effect of MMF itself.[18]

Primary Biliary Cholangitis

The use of LT for PBC has declined in recent decades, largely because of the widespread adoption of ursodeoxycholic acid (UDCA) and the introduction of effective second-line agents.[29] Graft survival in PBC remains excellent, with rates of 85–94% at 5 years and 81–90% at 10 years.[30, 31]

Approximately 20–30% of recipients develop recurrent primary biliary cholangitis (rPBC) within 10 years after transplantation.[32] The persistence of antimitochondrial antibodies and elevated immunoglobulin M (IgM) levels post-transplant limits their diagnostic utility for rPBC however ascendance of IgM could predict disease recurrence. Therefore, a liver biopsy is essential for confirmation.[21] The histopathological features mirror those of native PBC, including mononuclear cell infiltrates in the portal tracts, portal granulomas, bile duct injury and loss, and bile ductular proliferation. These findings must be carefully differentiated from immune-mediated injury to small bile ducts caused by rejection.[33]

Risk factors for rPBC include younger recipient age, onset of early post-transplant cholestasis, and use of certain immunosuppressive agents (Table 1).[30, 34, 35] Tacrolimus is associated with a higher risk of rPBC, whereas cyclosporine appears to confer a lower risk. However, the selection of calcineurin inhibitors does not seem to affect long-term graft or patient survival, supporting the ongoing use of tacrolimus after LT in patients with PBC.[36, 37] A multicenter study assessed the long-term effects of preventive UDCA therapy after LT in 941 patients transplanted for PBC, of whom 211 began UDCA (10–15 mg/kg/day) within the first two weeks post-transplant. Preventive UDCA use was associated with a reduced risk of rPBC and improved graft and patient survival.[38]

Primary Sclerosing Cholangitis

Patients who undergo transplantation due to PSC have survival rates of 90% at 1 year and 80% at 5 years after LT.[39, 40] However, PSC recurs in 20% to 25% of transplanted patients within 5 years.[41, 42] In a cohort of 306 PSC liver transplant recipients, the 5- and 10-year recurrence rates were 8.7% and 22.4%, respectively, with no significant difference between living and deceased donor transplants. In a European registry analysis of 29,902 liver transplants, long-term survival after living liver donor transplantation (LDLT) for AILD was generally favorable, but adult and pediatric PSC patients had higher mortality compared to donation after brain death (DBD) transplants. In adults diagnosed with PSC, LDLT has been correlated with an elevated risk of mortality due to disease recurrence and biliary complications. Additionally, the sex of the donor, specifically male, has been identified as a contributing factor to mortality.[43] Recurrent primary sclerosing cholangitis (rPSC) typically manifests 90 days post-transplant, characterized by ischemia and non-anastomotic biliary strictures. It is crucial to distinguish rPSC from ischemia, ABO incompatibility, cytomegalovirus infection, and chronic rejection. Notably, differentiation from chronic rejection is particularly important, as both conditions can present with ductopenia.

Table 1. Risk Factors for Recurrence of Autoimmune Liver Diseases After Liver Transplantation

Disease	Risk Factors for Recurrence
Autoimmune Hepatitis	Younger recipient age; high disease activity prior to LT (elevated IgG, high transaminases, severe inflammation in explant); donor–recipient sex mismatch; use of MMF; corticosteroid withdrawal
Primary Sclerosing Cholangitis	Younger or older recipient age; male sex; recurrent cholangitis before LT; high MELD score; cholangiocarcinoma; HLA DRB1*08 haplotype; IBD; older donor age; T-cell–mediated rejection; certain immunosuppressive regimens (anti-thymocyte globulin, tacrolimus, prolonged corticosteroid use)
Primary Biliary Cholangitis	Younger recipient age; early post-LT cholestasis; tacrolimus use (higher risk); absence of preventive UDCA therapy

LT: Liver transplantation; IgG: Immunoglobulin G; MMF: Mycophenate Mofetil; MELD: Model for end stage liver disease; IBD: Inflammatory bowel disease.

The histological characteristics of recurrent rPSC closely resemble those observed in primary sclerosing cholangitis (PSC) prior to transplantation. These characteristics include fibrous cholangitis, fibro-obliterative cholangitis with or without ductopenia, as well as portal, peri-portal, or bridging fibrosis, and cirrhosis.[44]

Multiple recipient, donor, and disease-related factors have been associated with increased risk of rPSC after liver transplantation. Recipient factors include younger or older age at LT, male sex, recurrent cholangitis before LT, high MELD score, cholangiocarcinoma, HLA DRB1*08 haplotype and the presence of inflammatory bowel disease (IBD).[45-48] While the literature presents inconsistent data regarding the efficacy of colectomy in preventing recurrent primary sclerosing cholangitis (rPSC), a recent meta-analysis has indicated a protective effect of colectomy against rPSC.[49] The evidence concerning the role of immunosuppressive regimens in rPSC is similarly contradictory. On one hand, the administration of anti-thymocyte globulin, tacrolimus, and extended corticosteroid therapy has been linked to rPSC, whereas the data on cyclosporine remain inconclusive.[16, 50-53] Conversely, reduced immunosuppression through single-agent therapy has also been associated with rPSC. Importantly, TCMR appears to be a significant precipitant of rPSC, contrasting with other liver transplant indications where it may facilitate long-term tolerance.[48] Nonetheless, tacrolimus monotherapy continues to be the cornerstone of maintenance immunosuppression in patients who have undergone transplantation for PSC.[54]

Immunosuppressive Strategies in Autoimmune Liver Diseases

Post-transplant immunosuppressive regimens for AILDs are designed to prevent both allograft rejection and disease recurrence while minimizing long-term toxicity (Fig. 1). Induction therapy in the early postoperative period typically consists of either basiliximab or high-dose corticosteroids, followed by tacrolimus-based maintenance. The choice and duration of adjunctive therapy depend on the underlying disease.

In AIH, triple-drug therapy is advised during the first year, then reduced to dual therapy for long-term maintenance,

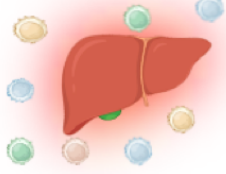
	Autoimmune Hepatitis	Primary biliary cholangitis	Primary sclerosing cholangitis
			
Induction Agent (First Week Post-LT)	Basiliximab IV: 2 × 20 mg at POD 0 & 4 OR Methylprednisolone IV: 500 mg at POD 0, taper over 4 days (250–125–80–40 mg)	Basiliximab IV: 2 × 20 mg at POD 0 & 4 OR Methylprednisolone IV: 500 mg at POD 0, taper over 4 days (250–125–80–40 mg)	Basiliximab IV: 2 × 20 mg at POD 0 & 4 OR Methylprednisolone IV: 500 mg at POD 0, taper over 4 days (250–125–80–40 mg)
Backbone Treatment	Tacrolimus (BID or OD, prolonged release) Target trough: 7–10 ng/mL (1st mo), 4–8 ng/mL (mo 1–12), 4 ng/mL (after 12 mo) Duration: Unlimited	Tacrolimus (BID or OD, prolonged release) Target trough: 7–10 ng/mL (1st mo), 4–8 ng/mL (mo 1–12), 4 ng/mL (after 12 mo) Duration: Unlimited	Tacrolimus (BID or OD, prolonged release) Target trough: 7–10 ng/mL (1st mo), 4–8 ng/mL (mo 1–12), 4 ng/mL (after 12 mo) Duration: Unlimited
Coadjuvant Therapy	Mycophenolate mofetil: 1000–2000 mg/d (1st year), then 250–500 mg BID long term OR Azathioprine: 1–1.5 mg/kg/d (1st year), then 50–75 mg OD long term OR Predniso(lo)ne: start 30 mg OD, taper over 1st year, maintain 5–10 mg OD long term Recommendation: Triple therapy in year 1, dual therapy long term	Mycophenolate mofetil: 1000–2000 mg/d, stop at 6–12 mo Azathioprine: 1–1.5 mg/kg/d, stop at 6–12 mo Predniso(lo)ne: start 30 mg OD, taper, stop at 3–6 mo Note: Long-term predniso(lo)ne not recommended	If IBD present: Azathioprine 1–1.5 mg/kg/d, stop at 6–12 mo (consider long-term if IBD active) If no IBD: Mycophenolate mofetil 1000–2000 mg/d, stop at 6–12 mo Predniso(lo)ne: start 30 mg OD, taper, stop at 3–6 mo Note: Long-term predniso(lo)ne not recommended

Figure 1. Suggested Immunosuppressive Regimens for Patients with AILD After Liver Transplantation.

AILD: Autoimmune liver disease; IBD: Inflammatory bowel disease; LT: Liver transplantation; POD: Postoperative day.

with monotherapy discouraged due to the high risk of recurrence. In PBC, adjunctive agents are used only short term, corticosteroids are withdrawn early, and tacrolimus monotherapy is permissible; prophylactic UDCA should be continued because of its proven benefits in this population. In PSC, azathioprine may be maintained long term in patients with active inflammatory bowel disease, whereas others receive only short-term adjunctive therapy; corticosteroids are tapered early, and tacrolimus monotherapy is an acceptable maintenance strategy.

Management of recurrent autoimmune liver diseases after transplantation is tailored to the specific condition. rAIH is typically treated by intensifying immunosuppression, most often through reintroduction or escalation of corticosteroids, sometimes in combination with everolimus or mycophenolate mofetil, with prolonged maintenance therapy to minimize further recurrence. Primary management of rPBC involves the long-term administration of UDCA, which has been shown to improve biochemical parameters and may potentially decelerate histologic progression. In cases where additional treatment is necessary, second-line agents such as bezafibrate or obeticholic acid may be incorporated. Baseline immunosuppression is generally unchanged unless concomitant rejection is present. rPSC management is largely supportive, involving endoscopic dilation or stenting of dominant strictures, antibiotic treatment of recurrent cholangitis, and careful optimization of immunosuppression to prevent further injury. No proven medical therapy halts rPSC progression, and severe or progressive cases may necessitate re-transplantation.[54] Treatment of chronic rejection is similar to that for other disease etiologies and involves increasing tacrolimus trough levels, along with the addition of a second-line agent, typically everolimus or MMF.[55]

Conclusion

AILDs are important indications for liver transplantation and carry higher risks of rejection and recurrence than non-immune etiologies. Optimizing outcomes relies on tailored immunosuppression, disease-specific adjunctive therapies, and vigilant long-term follow-up to detect and manage recurrence or chronic rejection early.

Disclosures

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