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REVIEW

Liver Disease in Older Adults: Diagnosis, Management, and Transplantations

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Abstract

Purpose of Review To summarize age-specific considerations in the diagnosis and management of liver disease in adults aged 65 years and older, with emphasis on cirrhosis, liver transplantation, frailty, and cognitive impairment.

Recent Findings Age alters the performance and tolerability of commonly used diagnostic and therapeutic strategies. Age-adjusted interpretation of serum fibrosis tests, sequential use of elastography, careful titration of cirrhosis therapies, and routine frailty assessment can reduce avoidable harm. Transplant outcomes in carefully selected older recipients remain acceptable, and chronological age alone should not determine candidacy.

Summary The care of the older adults with liver disease should integrate disease severity with physiologic reserve, comorbidity, function, medication safety, and cognition. A structured evaluation is particularly important when hepatic encephalopathy overlaps with delirium or dementia.

Keywords Elderly · Hepatic encephalopathy · Cirrhosis · Dementia · Liver transplantation

Introduction

Claims-based estimates place liver disease prevalence at 5.07% among Medicare beneficiaries, compared with 1.45% among privately insured enrollees of all ages [1]. Among Medicare beneficiaries, prevalence increased by more than 50% between 2015 and 2021 [1]. Globally, MASLD prevalence in the elderly (aged 65–89 years) was estimated at 228 million cases in 2019, a 42% increase from 2010 [2]. The global burden of liver cirrhosis in older adults has similarly risen in absolute numbers, though age-standardized death rates have declined modestly [3]. This epidemiologic shift is driven by the rising prevalence of metabolic syndrome, continued consequences of historical hepatitis C virus (HCV) infection, and increasing alcohol-related liver disease [4, 5].

Aging alters hepatic physiology, reducing liver volume, hepatic blood flow, and drug metabolism, while increasing susceptibility to hepatic steatosis [4]. These changes have implications across the continuum of liver disease, from screening and diagnosis through management of advanced disease and consideration of liver transplantation.

The current review describes the diagnosis of clinically important liver disease, cirrhosis and complications, liver transplantation and then focuses specifically on frailty, polypharmacy, and cognitive impairment in older adults with liver disease.

Diagnostic Approaches in Older Adults: Noninvasive Tests, Imaging and Liver Biopsy

Noninvasive Tests and Age-Related Limitations

Noninvasive tests (NITs) have become the cornerstone of liver fibrosis assessment, reducing the need for liver biopsy. The Fibrosis-4 (FIB-4) index calculated using age, AST, ALT, and platelet count is one of the most validated first-line screening tools [5, 6]. However, because age is a component of the FIB-4 formula, scores are systematically higher in older patients, leading to reduced specificity and a high false-positive rate in patients aged 65 years and older [7].

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McPherson et al. demonstrated that FIB-4 specificity for advanced fibrosis dropped to 35% in patients aged ≥ 65 years using standard cutoffs, compared with 70–80% in middle-aged adults [7]. They derived and validated age-adjusted cutoffs: a FIB-4 threshold of 2.0 (rather than 1.3) to exclude advanced fibrosis in patients ≥ 65 years, maintaining sensitivity at 77% while improving specificity to 70% [7]. An alternative strategy used is to cap the age at 65 years regardless of the real age beyond this number [8]. Similarly, the NAFLD Fibrosis Score (NFS) requires an adjusted lower cutoff of 0.12 in this population, as the standard cutoff yielded a specificity of only 20% [7, 9]. These age-adjusted thresholds are now endorsed by the AGA clinical care pathway and AASLD practice guidance for MASLD [10, 11].

Despite these adjustments, all serum-based NITs share the limitation of poor positive predictive value, particularly in older adults, in whom comorbidities such as diabetes and cardiovascular disease may independently affect platelet counts and transaminase levels [6, 9]. The Enhanced Liver Fibrosis (ELF) panel, a proprietary test measuring hyaluronic acid, TIMP-1, and N-terminal procollagen III peptide, may offer advantages in this population, as it is not age-dependent and has shown prognostic value for liver-related events [10, 12].

Imaging-Based Assessment

Vibration-controlled transient elastography (VCTE) serves as a valuable second-line test when FIB-4 is elevated or indeterminate. A liver stiffness measurement (LSM) ≥ 15 kPa identifies cirrhosis with 95.5% specificity, while LSM ≥ 10 kPa achieves 88% negative predictive value [6]. Sequential testing using FIB-4 followed by VCTE can achieve posterior probabilities of cirrhosis exceeding 89% [6]. Importantly, VCTE performance appears similar in lean and obese patients, though false-positive elevations can occur in older adults with congestive heart failure or hepatic congestion, conditions that are more prevalent with aging [6, 13].

Magnetic resonance elastography (MRE) offers higher technical success rates in patients with elevated BMI and is less affected by body habitus [13]. However, cost and access limitations restrict its use as a primary screening tool. MRE may be considered as a confirmatory test, particularly when VCTE results are discordant with clinical findings [13].

Liver Biopsy in the Older Patient

Liver biopsy remains the reference standard for fibrosis staging and is essential for distinguishing steatohepatitis from simple steatosis, evaluating coexisting pathology, and diagnosing conditions where NITs lack validation [6, 13]. However, biopsy carries procedural risks that may be amplified in older adults due to coagulopathy, thrombocytopenia, comorbid anticoagulation use, and reduced physiological reserve. The trend toward noninvasive assessment has been particularly beneficial for older patients, and current societal guidance recommends a sequential NIT-based approach, reserving biopsy for situations in which the diagnosis remains uncertain or histologic detail is needed to guide therapy [10, 11, 13].

Comparative Considerations: Older vs. Younger Adults

In younger adults, both FIB-4 and NFS perform poorly for identifying advanced fibrosis (AUROC 0.52–0.60), while in the 35–65 year range, diagnostic accuracy is highest (AUROC 0.77–0.84) [7]. In patients ≥ 65 years, the diagnostic accuracy of serum-based NITs is preserved (similar AUROCs), but specificity declines substantially without age-adjusted thresholds [7]. Imaging-based modalities such as VCTE and MRE are less influenced by age and may therefore be preferred second-line tools in older populations [6, 13].

Management of Compensated and Decompensated Cirrhosis in Older Adults

Compensated Cirrhosis

Compensated cirrhosis or the entity known as compensated advanced chronic liver disease (cACLD) carries a relatively favorable prognosis when progression to decompensation is prevented. Portal hypertension is the major driver of the transition from compensated to decompensated disease, and a hepatic venous pressure gradient ≥ 10 mmHg defines clinically significant portal hypertension (CSPH) [14]. Baveno VIII further expand upon the definition of CSPH with the use of clinical imaging surrogates or NITs such as using including liver stiffness by VCTE (≥ 20–25 kPa suggesting CSPH) combined with platelet count [15].

The PREDESCI trial and subsequent data have demonstrated that nonselective beta-blockers (NSBBs) prevent decompensation (ascites and variceal bleeding) in patients with cACLD and CSPH, primarily by reducing the risk of ascites [14]. In a 3-year randomized trial of 201 patients, NSBBs reduced the risk of decompensation or death (16% vs. 27% with placebo) [6]. Carvedilol has emerged as the preferred NSBB due to its additional anti-alpha-1 adrenergic effects and greater portal pressure reduction [6].

In older adults, NSBBs require careful titration because of age-related hemodynamic changes, the higher prevalence of conduction abnormalities and orthostatic hypotension,

and polypharmacy. Carvedilol's vasodilatory properties may increase fall risk in frail older patients, and heart rate targets may need adjustment given lower baseline heart rates with aging.

Etiological treatment remains the most effective strategy for halting progression: HCV eradication with direct-acting antivirals achieves sustained virologic response rates exceeding 95% across all age groups and can lead to cirrhosis regression [14]. For MASLD, lifestyle modifications (caloric restriction, 150 min of weekly exercise) form the foundation, and resmetirom and semaglutide are now FDA-approved for noncirrhotic MASH with F2-F3 fibrosis [14, 16]. Alcohol cessation is critical in alcohol-related liver disease, and naltrexone is the preferred first-line agent for moderate-to-severe alcohol misuse in older adults under careful supervision [4].

Decompensated Cirrhosis

Decompensated cirrhosis (dACLD), defined by the presence of ascites, variceal hemorrhage, or hepatic encephalopathy, carries a markedly worse prognosis: median survival is approximately 1 year after the onset of ascites or encephalopathy [6]. Management of decompensating events in older adults presents unique challenges.

Ascites First-line therapy is sodium restriction and diuretics. The combination of an aldosterone antagonist and a loop diuretic is more effective than sequential initiation (resolution in 76% vs. 56%) and is associated with lower rates of hyperkalemia [6]. However, in older patients, overly aggressive diuresis can rapidly precipitate acute kidney injury, hyponatremia, and hemodynamic instability [4]. Careful monitoring of renal function and electrolytes is essential, and lower starting doses with slower titration are prudent. Therapeutic paracentesis with albumin replacement is used for refractory or tense ascites. Transjugular intrahepatic portosystemic shunt (TIPS) is increasingly considered earlier in the disease course, though older age and associated cardiac and renal comorbidities may increase procedural risk [17].

Variceal Hemorrhage Similarly to for ascites, pre-emptive TIPS for high-risk variceal bleeding (Child-Pugh score 10–13, or 8–9 with active bleeding) has become standard of care at many centers and improves mortality [17]. Age alone should not preclude TIPS placement, but cardiovascular assessment is essential given the hemodynamic consequences of reversing the portosystemic shunting acutely post-TIPS.

Hepatic Encephalopathy The main challenge (mentioned below) is the diagnosis of HE in patients who are prone to delirium and cognitive impairment due to multiple other causes. In those with suspected confusion due to HE, inpatient management should be empirically treated for OHE utilizing PO lactulose titrated to 3–4 bowel movements per day, pending evaluation of alternative etiologies [18]. Many of the known HE precipitants (i.e., hypokalemia, renal failure, gastrointestinal bleed) are linked to ammonia production and/or absorption. Other factors (i.e., infection, sedative use, electrolyte disturbance, constipation) may potentiate the neurotoxic effects of ammonia, alter the permeability of the BBB, or work independently to facilitate HE. Dehydration from excessive bowel movements from over-use of therapy (laxatives) is another potential cause of acute HE. A thorough history obtained from the patient or family/friends can help guide etiology. To prevent recurrence after discharge, lactulose remains first-line therapy, reducing mortality (8.5% vs. 14% with placebo) and recurrent episodes (25.5% vs. 46.8%) [6]. However, lactulose use can be challenging in older adults due to diarrhea-induced dehydration, electrolyte disturbances, and falls [4]. Rifaximin is second-line therapy for refractory cases. Emerging adjunctive therapies, including L-ornithine L-aspartate and albumin, require further study, particularly in this population [17].

Hepatorenal Syndrome Terlipressin combined with albumin improved reversal of hepatorenal syndrome (39% vs. 18% with placebo) [6]. In older adults, terlipressin carries heightened risk of cardiovascular ischemic events, requiring careful patient selection and monitoring [19].

Age-Specific Considerations

Comorbid conditions prevalent in older adults including diabetes, cardiovascular disease, chronic kidney disease, and polypharmacy accelerate progression to decompensation and complicate management [4]. Hepatocellular carcinoma surveillance with ultrasound and alpha-fetoprotein every 6 months should be maintained in older adults who are candidates for curative or palliative therapy [4].

Liver Transplantation in the Older Adult

Evolving Age Criteria

The proportion of liver transplant recipients aged ≥ 65 years increased from 1.6% in 1988 to 21% in 2023 [4]. The AASLD practice guidelines state that physiological eligibility for liver transplant should not be determined by chronological age, and age ≥ 70 years is not considered a contraindication [20, 21].

Outcomes by Age

Five-year graft survival for recipients ≥ 65 years was 74.0% in 2020, compared with > 75% for younger age categories-a relatively modest difference [4]. A single-center study of 999 recipients found no differences in mortality or graft failure at 1, 3, and 5 years between patients ≥ 70 and < 70 years, though the older cohort had significantly longer hospital stays (25.5 vs. 14.0 days) [22]. Mousa et al. reported 5-year survival of 70.8% in septuagenarian recipients versus 80.7% in recipients in their 60s, with no incremental negative effect of age between 70 and 75 years [23]. In a propensity-score matched analysis, patients over 70 achieved post-transplant outcomes comparable to younger patients when matched for MELD score and cirrhosis etiology [24].

However, longer-term outcomes are less favorable: 10-year survival in elderly recipients (43.6%) was substantially lower than in younger groups (67.6%), reflecting competing causes of mortality [23]. Each 10-year increment in recipient age was associated with increased mortality risk (HR 1.20) in the Adult-to-Adult Living Donor Liver Transplantation Cohort [21].

Donor-Recipient Age Matching

Donor-recipient age matching may inform graft allocation. Among older recipients (> 65–70 years), graft survival is relatively stable regardless of donor age, whereas younger recipients show steep declines in graft survival with increasing donor age [25]. This suggests that older donor grafts may be acceptably allocated to older recipients without compromising outcomes.

When Age Might Preclude Transplantation

While no absolute age cutoff exists, the decision to list an older patient must incorporate a comprehensive assessment of physiological reserve, cardiovascular risk, functional status, malignancy risk, and projected transplant-related survival benefit [21]. Transplant-related survival benefit the incremental survival gained from transplantation rather than remaining on the waitlist provides a more complete framework than post-transplant survival alone [21]. Mousa et al. demonstrated that survival of transplant recipients, regardless of age, markedly surpassed that of patients denied transplantation, though it remained 20–30% lower than the expected survival of the general age-matched US population [23].

Post-transplant considerations in older recipients include higher rates of hepatic artery thrombosis, cardiovascular and renal complications, postoperative delirium, and greater transfusion requirements [4]. Immunosenescence may reduce the risk of acute rejection while increasing susceptibility to infection; in selected older recipients, this balance may support earlier reduction of immunosuppression [4].

Frailty and Liver Disease

Definition and Assessment

Frailty is a multidimensional state of vulnerability with physical, cognitive, nutritional, and social domains. In cirrhosis, sarcopenia and frailty are driven by catabolic metabolism, poor protein intake, systemic inflammation, deconditioning, fluid retention, and encephalopathy [4]. Three validated frailty instruments are used in cirrhosis: the Liver Frailty Index (LFI), Fried Frailty Index, and Clinical Frailty Scale [4].

Sarcopenia can be assessed via the skeletal muscle index at L3 on CT, bioelectrical impedance analysis, DEXA, and mid-arm muscle circumference, though each has limitations in patients with fluid retention [4]. Hand grip strength shows an inverse correlation with waitlist mortality [4].

Impact on Outcomes

Pretransplant frailty, as measured by the LFI, is associated with a nearly 2-fold increased risk of waitlist mortality (sub-HR 1.82; 95% CI 1.31–2.52), independent of MELD, ascites, and encephalopathy [26]. Post-transplant, frailty confers a 2-fold increased risk of death (HR 2.13; 95% CI 1.39–3.26) and greater healthcare utilization [21]. Changes in frailty over time are equally informative: each 0.1-unit worsening in LFI over 3 months doubled the hazard of waitlist mortality [26].

Frailty and Age

Even before cirrhosis develops, sarcopenia, which is especially prevalent in older adults owing to overlapping age-related and disease-related muscle loss is an independent risk factor for progression of MASLD to advanced fibrosis, cirrhosis, and liver-related mortality, mediated by shared mechanisms of insulin resistance, chronic inflammation, and dysregulated hepatokine–myokine crosstalk [27]. Cirrhosis-related sarcopenia is exacerbated in older individuals, compounding age-related loss of muscle mass and function [4]. This overlap increases fall risk, loss of independence, and hospitalization. Screening for frailty should be integrated into routine evaluation of older patients with cirrhosis, particularly in the transplant evaluation setting [4].

Frailty and Transplant Candidacy

Frailty is not an absolute contraindication to liver transplantation. US multicenter data demonstrate a net survival benefit from transplantation across all levels of frailty

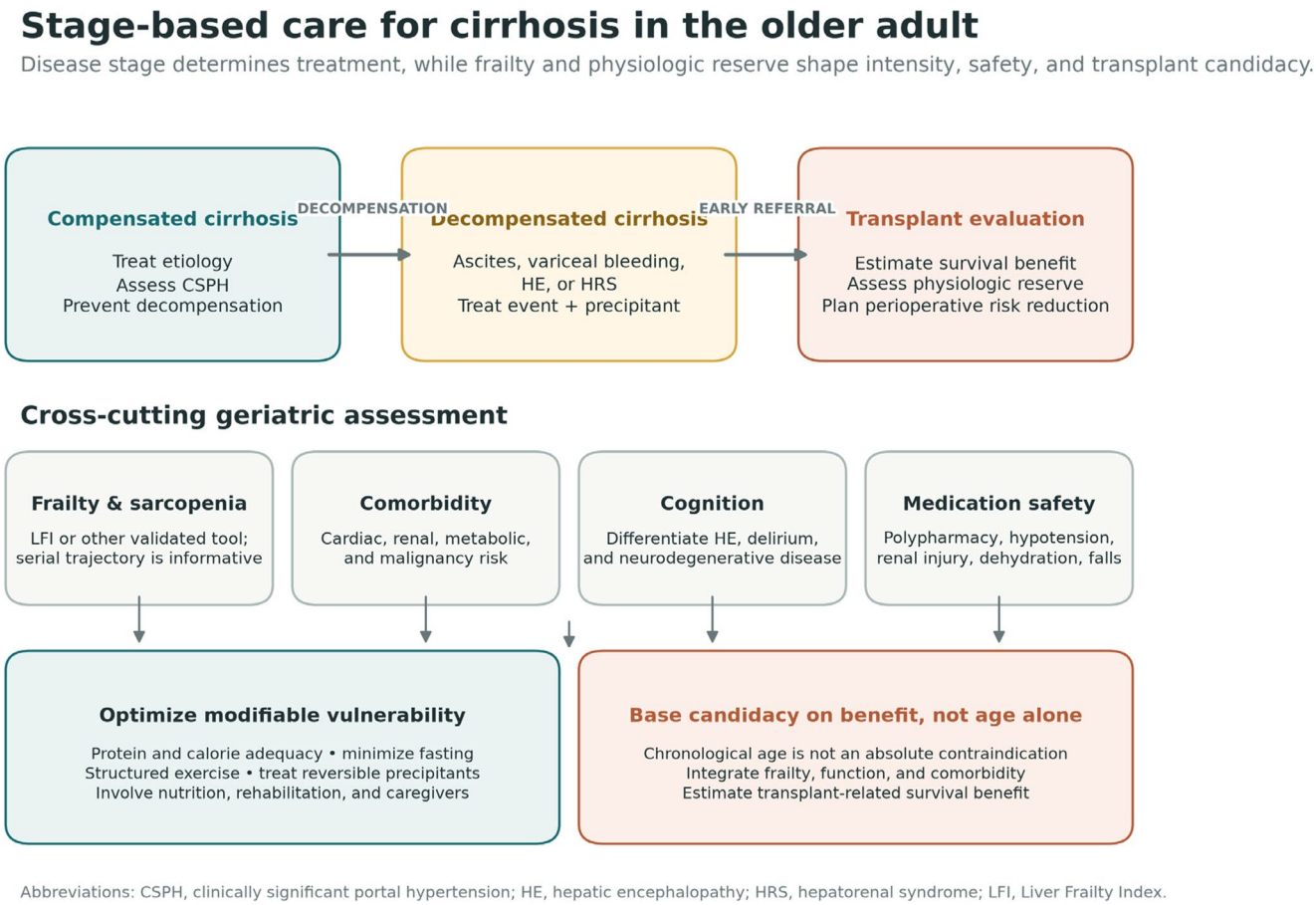

Fig. 1 Stage-based care for cirrhosis in the older adult. Management should follow disease stage while incorporating frailty, sarcopenia, comorbidity, cognition, and medication safety across the continuum of care. Transplant candidacy should be based on physiologic reserve and expected survival benefit rather than chronological age alone. This figure was generated with the assistance of Perplexity AI tool, with all output was reviewed for scientific and factual accuracy and edited by the authors who take full responsibility for the final content of the figure

measured by the LFI [21]. However, only 40% of patients achieve "robustness" at 1 year post-transplant, highlighting an unmet need for prehabilitation interventions [21]. Optimization strategies include protein intake of 1.5–2 g/kg/day, caloric intake of 30–40 kcal/kg/day, minimizing fasting intervals with nighttime snacks, glycemic optimization, and structured exercise programs [4]. A multidisciplinary approach involving nutrition, physiotherapy, and psychotherapy is recommended [4, 28] (Figure 1) [4, 21, 26, 28].

Hepatic Encephalopathy, Dementia, and other causes of cognitive impairment

The Diagnostic Challenge

Hepatic encephalopathy (HE) in older patients poses one of the most difficult diagnostic challenges in hepatology. Clinicals must have heightened clinical awareness: dementia and HE frequently co-occur, and HE, especially its minimal/covert form with insidious progression, can masquerade as dementia when cirrhosis goes unrecognized, delaying potentially reversible HE-directed therapy [8, 29].

Overt HE classically presents as acute delirium with disorientation and asterixis. However, covert HE (CHE) presents with subtle attention deficits, sleep disturbance, irritability, and mild confusion that are easily attributed to aging, mild cognitive impairment (MCI), or early dementia [4, 30].

Several features contribute to this diagnostic overlap. The evolution from CHE to overt HE may resemble the gradual cognitive decline of neurodegenerative disease, particularly when cirrhosis is unrecognized [4]. Extrapyramidal manifestations of HE including parkinsonism, chorea, and spastic paraparesis can also resemble Lewy body dementia or Parkinson's disease dementia [4, 30]. In patients with pre-existing dementia, superimposed HE may present as delirium and can be difficult to recognize [4, 30].

Distinguishing Features

Several features may help differentiate HE from primary dementia. HE is fundamentally a disorder of attention and arousal, whereas dementia primarily affects memory, language, and executive function [4]. The hallmark of HE is fluctuation, waxing and waning mental status whereas dementia follows a gradual, progressive course [4]. Asterixis, when present, strongly supports HE. The Confusion Assessment Method (CAM) criteria of acute onset or fluctuating course, inattention, disorganized thinking, and altered consciousness can help identify delirium due to HE, although the "attenuated delirium syndrome" variant of mHE may meet only some CAM criteria [4].

No single feature reliably distinguishes the two conditions. Both HE and dementia can present with apathy, psychomotor slowing, and impaired executive function. Extrapyramidal features overlap between HE, Lewy body dementia, and Parkinson's disease dementia [4]. Chronic or recurrent HE may also cause persistent cognitive deficits that do not fully reverse, further blurring the boundary with neurodegenerative disease.

Screening Tools

An analysis of 286 patients with cirrhosis and cognitive complaints found that more than 50% referred for presumed HE did not have mHE, with differential diagnoses including MCI, dementia, mood disorders, PTSD, and obstructive sleep apnea [18]. This underscores the need for systematic cognitive assessment rather than empiric HE treatment.

The EncephalApp Stroop test, available as a smartphone application, and the Simplified Animal Naming Test (S-ANT1) provide practical screening for mHE; Quick-Stroop and S-ANT1 may also help distinguish mHE from other causes of cognitive change [4, 30]. If screening is abnormal, detailed caregiver history regarding alertness changes, symptom fluctuation, and functional decline should follow [4]. For patients with known dementia, the modified Richmond Agitation and Sedation Scale (m-RASS) provides a simple observation-based assessment of alertness that can identify superimposed delirium (potentially HE) without requiring patient questioning [4, 30].

A Practical Approach

In older patients with cirrhosis and cognitive symptoms, a systematic evaluation should include [18, 30]:

- assessment for precipitating factors of HE (infection, GI bleeding, constipation, medication effects, electrolyte disturbances).
- a validated screening tool such as S-ANT1 or EncephalApp Stroop.
- detailed medication review to exclude pharmacologic contributors.
- evaluation for obstructive sleep apnea.
- if dementia is suspected, formal neuropsychological testing.

Empiric lactulose and rifaximin should not be given to every older patient with cirrhosis and cognitive symptoms. Unnecessary treatment may cause adverse effects and delay identification of the actual disorder [18].

Conclusions

Care of older adults with liver disease should be guided by physiological reserve rather than chronological age alone. Age-adjusted fibrosis assessment, cautious management of cirrhosis complications, and routine evaluation of frailty can individualize treatment and transplant decisions. When cognitive symptoms arise, structured assessment is essential to distinguish hepatic encephalopathy from dementia and other causes before therapy is initiated.

Annotated Key References

- Brown CL, Bajaj JS. Considerations in the diagnoses and management of hepatic disease in older adults. *American Journal of Gastroenterology.* 2025.
  - This is relevant recent reference for the manuscript's overarching focus on liver disease in adults aged 65 years and older. It provides a geriatric-centered framework for adapting diagnosis and treatment to changes in physiology, comorbidity burden, polypharmacy, frailty, and cognitive vulnerability; all of which are topics discussed in this review.
- Tapper EB, Parikh ND. Diagnosis and management of cirrhosis and its complications: a review. *JAMA.* 2023;329(18):1589–1602.
  - This high-impact review supports the manuscript's discussion of compensated and decompensated cirrhosis, including portal hypertension, ascites, variceal bleeding, hepatic encephalopathy, and hepatorenal syndrome. It is especially valuable as a broad, clinically practical evidence that the manuscript adds the age-specific considerations needed for older adults.

- Bajaj JS, Silvey SG, Rogal S, et al. Undiagnosed Cirrhosis and Hepatic Encephalopathy in a National Cohort of Veterans With Dementia. *JAMA Netw Open*. 2024;7(1):e2353965.
  - Importance of looking for HE as an alternative diagnosis or contributor towards cognitive impairment in patients being treated as dementia. This was across national and local cohorts with confirmation and validation.
- Dove L, Chadha RM, Lai JC, et al. AASLD AST practice guideline on adult liver transplantation: candidate evaluation. *Hepatology.* 2025.
  - This guideline is a key reference regarding transplantation because it frames candidacy around physiologic reserve, comorbid conditions, functional capacity, and expected survival benefit rather than chronological age alone. It also reinforces the manuscript's emphasis on frailty assessment and individualized transplant evaluation in older candidates.

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Data Availability No datasets were generated or analysed during the current study.

Declarations

Competing Interests The authors declare no competing interests.
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