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REVIEW

Biological Selection in Liver Transplant Oncology: Current Indications and Limits for Hepatocellular Carcinoma, Cholangiocarcinoma and Colorectal Liver Metastases

Ali MUHTAROĞLU,[1] Kayhan ÖZDEMİR,[2] Berkan ACAR,[3] Elif YILMAZ,[4] Murat AKALIN,[5] Yavuz ÇEKİÇ,[6] Fatih ALTINTOPRAK[2]

[1]Department of General Surgery, Private Adatip Hospital, Sakarya-*Türkiye*
[2]Department of General Surgery, Sakarya University Faculty of Medicine, Sakarya-*Türkiye*
[3]Department of General Surgery, Giresun University Faculty of Medicine, Giresun-*Türkiye*
[4]Department of General Surgery, Private Umut Hospital, Ordu-*Türkiye*
[5]Department ofGeneral Surgery, Bakırçay University Faculty of Medicine, Çiğli Training and Research Hospital, İzmir-*Türkiye*
[6]Department of General Surgery, School of Surgery NHSE, Yorkshire and Humber Deanery, Leeds-*England*

SUMMARY

Liver transplant oncology has moved beyond the blunt expansion of size-based criteria. The central question now is whether tumour behaviour during treatment can identify the uncommon patient whose disease remains transplantable. This review examines hepatocellular carcinoma, perihilar and intrahepatic cholangiocarcinoma, and colorectal liver metastases, with emphasis on the evidence and policy shifts most relevant to current practice. In hepatocellular carcinoma, downstaging is best understood as a biological stress test rather than a technical bridge, and alpha-fetoprotein trend, radiological response, and time under observation now refine morphology-based thresholds. In perihilar cholangiocarcinoma, the lesson is different but equally clear: Transplantation succeeds when diagnosis, neoadjuvant therapy, staging, and operative discipline are delivered as a protocol. In intrahepatic cholangiocarcinoma, transplantation has become credible in very early cirrhotic tumours and in exceptionally selected liver-limited disease with durable treatment response, although the evidence remains centre-specific. Colorectal liver metastases mark the sharpest frontier, where comparative data have made transplantation intellectually defensible but not routine. Across these indications, the common denominator is biological selection, transparent reporting of the denominator, and careful stewardship of a scarce graft. The field will advance less by indiscriminately broadening criteria than by more precisely defining which cancers, in which biological states, genuinely justify transplantation.

Keywords: Cholangiocarcinoma; colorectal carcinoma; hepatocellular carcinoma; liver transplantation; metastasis; organ transplantation.

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INTRODUCTION

The old language of 'expanded criteria' no longer describes where transplant oncology actually stands. It suggests that progress comes mainly from relaxing limits that were once regarded as fixed. In reality, the field has advanced by becoming more selective, not less. Size and number still matter, but they now sit

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Dr. Ali MUHTAROĞLU
Özel Adatip Hastanesi,
Genel Cerrahi Kliniği,
Sakarya-Türkiye
E-mail: alimuhtarogluu@gmail.com

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beside alpha-fetoprotein, radiological response, interval observation, molecular profile and the broader tempo of disease.[1–6]

It is useful to separate mature indications from emerging ones. Hepatocellular carcinoma remains the benchmark indication and the reference point against which all newer oncological indications are judged. Perihilar cholangiocarcinoma has moved into the category of an established but protocol-dependent indication. Intrahepatic cholangiocarcinoma and colorectal liver metastases sit further forward on the curve: Both are now clinically real, neither can be dismissed as experimental in the old sense, but both still demand tighter selection and more transparent reporting than hepatocellular carcinoma (Table 1).[5–23]

Policy has started to reflect this change. In the United States, the 2025 guidance that brought hepatocellular carcinoma, intrahepatic cholangiocarcinoma and colorectal liver metastases together under adult transplant-oncology review did more than tidy up administration. It acknowledged that transplant oncology can no longer be governed by hepatocellular carcinoma rules alone. The practical question is no longer whether liver transplantation can cure selected hepatic malignancy. It plainly can. The harder question is which tumours justify a scarce graft, under what evidential threshold, and with what obligation to report denominator data as carefully as headline survival.[5,6,10,16,17,23]

SEARCH STRATEGY AND SCOPE

This narrative review was designed to answer a practical transplant question: What now makes a liver cancer biologically transplantable? PubMed/MEDLINE, contemporary consensus statements, and current allocation guidance were reviewed, with an emphasis on prospective series, multicentre cohorts, policy documents, and higher-quality reviews that directly inform listing practice. The aim was not exhaustive historical coverage, but a clinically useful synthesis of the evidence most likely to change selection decisions today.

HEPATOCELLULAR CARCINOMA: THE BENCHMARK FOR BIOLOGICAL SELECTION

Hepatocellular carcinoma remains the model through which the field learned to think about curative transplantation in cirrhosis. Milan criteria remain foundational, not because they capture tumour biology perfectly, but because they created a reproducible oncological threshold.[1] UCSF criteria showed that modest expansion beyond Milan could preserve acceptable post-transplant outcomes,[2] while later models such as Metroticket 2.0 and the alpha-fetoprotein model made it clear that morphology alone was too blunt an instrument.[3,4] The 2024 ILTS-ILCA consensus captures the contemporary position well: Size and number still matter, but they should now be

Table 1 Current position of liver transplantation across the major transplant-oncology indications

Disease	Current position	Selection essentials	Representative clinical message
Hepatocellular carcinoma	Mature and established indication	Milan/T2 disease or protocol-based downstaging; alpha-fetoprotein trend; radiological response; interval observation; no extrahepatic spread or macrovascular invasion	Successful downstaging can yield 5-year post-transplant survival in the mid-70% range, but recurrence rises when tumour burden extends well beyond standard limits.
Perihilar cholangiocarcinoma	Established, but only in protocol-driven programmes	Unresectable hilar disease; approved neoadjuvant chemoradiation pathway; operative staging; no transperitoneal biopsy; strict exclusion of metastatic spread	In expert centres, 5-year survival of around 65%- 82% is achievable. Results deteriorate sharply when the protocol is diluted.
Intrahepatic cholangiocarcinoma	Emerging and deliberately constrained	Very early cirrhotic tumours or exceptionally selected liver-limited unresectable disease with durable response; prolonged stability on therapy; no extrahepatic disease	Very early disease can achieve 5-year survival around 65%-73%; selected locally advanced disease has shown promising 5-year survival, but evidence remains limited and centre-specific.
Colorectal liver metastases	Emerging indication in highly selected patients	Liver-only disease; resected primary; sustained response to first-line chemotherapy; favourable molecular profile; low carcinoembryonic antigen; adequate interval from diagnosis; no progression or extrahepatic spread	SECA-II and TransMet show survival that is difficult to match with non-transplant therapy alone, but recurrence is common, and selection must remain ruthless.

T2, OPTN stage T2 hepatocellular carcinoma.

interpreted alongside alpha-fetoprotein, treatment response and time under observation.[5]

In practice, downstaging is best understood as a biological stress test rather than a technical manoeuvre. Locoregional therapy does not simply shrink tumours; it reveals which tumours continue to behave in a transplantable manner when exposed to treatment and time. Long-term UCSF data, the prospective MERITS-LT experience and subsequent multicentre series all point in the same direction. When entry criteria, response thresholds, and observation intervals are met, successful downstaging can achieve post-transplant survival close to that observed in patients who began treatment according to conventional criteria.[24–28]

The same literature also clarifies the limits of expansion. Xu et al.[27] reported in a 2025 national multicentre prospective cohort that successful downstaging reached 82% within UNOS downstaging criteria but only 66% in all-comers beyond those limits. Once transplantation occurred, post-transplant survival was similar, but recurrence and intention-to-treat survival worsened as pre-treatment burden moved further beyond standard boundaries. Downstaging can therefore extend access responsibly, but only up to a point.[25–28]

Several practical conclusions follow. Alpha-fetoprotein trend is more informative than a single value. Waiting time is not merely an administrative burden; it is an opportunity to reveal aggressive biology. Choice of locoregional therapy matters, but the larger point is that response history matters more than the device used. Programmes that treat downstaging as a technical gateway rather than a biological filter risk selecting the very tumours most likely to recur.[24–28]

The next pressure point concerns systemic therapy, particularly immune checkpoint inhibition. Modern hepatocellular carcinoma management increasingly involves programmed death-1 or programmed death-ligand 1 blockade before referral, which means transplant teams now encounter candidates previously exposed to agents capable of provoking allograft rejection. The available evidence remains heterogeneous and retrospective. Some patients have proceeded safely to transplantation after washout, but acute rejection and graft loss are well documented, and no universally accepted safe interval can yet be defended.[5,29]

PERIHILAR CHOLANGIOCARCINOMA: SUCCESS DEPENDS ON THE WHOLE PROTOCOL

Perihilar cholangiocarcinoma is the clearest example of transplantation succeeding because an entire protocol works, not because a single operation does. Historic results were poor when patients were taken directly to transplantation without rigorous diagnosis, neoadjuvant therapy, operative staging or exclusion of occult spread. The Mayo experience changed that narrative. Heimbach et al.[7] first reported excellent survival in unresectable perihilar cholangiocarcinoma managed with neoadjuvant chemoradiation followed by transplantation. Rea et al.[8] then showed that protocol-based transplantation could outperform resection in selected patients, with 5-year survival around 82%. In the multicentre United States series reported by Murad et al.,[9] 5-year recurrence-free survival reached 65%, despite dropout during neoadjuvant treatment.

These data explain why perihilar cholangiocarcinoma should now be regarded as an established indication in expert centres. They also explain why casual enthusiasm is dangerous. Good results depend on the whole pathway: Accurate diagnosis, careful biliary management, disciplined neoadjuvant therapy, operative staging and strict exclusion of metastatic disease. Put bluntly, the protocol is part of the treatment.[7–12]

Current policy captures that reality. Under OPTN policy, standardised exception access for perihilar cholangiocarcinoma depends on management under an approved written protocol incorporating neoadjuvant therapy and operative staging. Diagnostic support may come from a malignant-appearing hilar stricture together with positive cytology or biopsy, a CA19-9 concentration above 100 U/mL in the absence of cholangitis, aneuploidy, or a hilar mass smaller than 3 cm. Transperitoneal biopsy should be avoided because of the recognised risk of tumour seeding.[10]

What matters in practice is not merely that transplantation works for perihilar cholangiocarcinoma, but why it works. The ideal candidate is not discovered on the day of transplantation; the ideal candidate is shaped by months of disease control, protocol adherence and repeated failure of the tumour to declare systemic behaviour. Centres that bypass one or more of these steps tend to recreate the disappointing results of the pre-protocol era.[7–12]

INTRAHEPATIC CHOLANGIOCARCINOMA: CREDIBLE, BUT STILL DELIBERATELY CONSTRAINED

If perihilar cholangiocarcinoma is now established, intrahepatic cholangiocarcinoma remains the field's most interesting unsettled indication. The older lit-

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erature was difficult to interpret because it mixed tiny incidental tumours found in explants with large, biologically aggressive cancers transplanted without a coherent selection strategy. That mixture obscured the fact that intrahepatic cholangiocarcinoma is not one transplant population.[11,12]

Two routes to transplantation have now emerged. The first concerns very early intrahepatic cholangiocarcinoma in cirrhotic patients, usually solitary lesions no larger than 2 cm. In this setting, Sapisochin et al.[30,31] showed that survival could be substantially better than the historical literature suggested, with 5-year survival ranging from 65% to 73%. Those studies mattered not because they proved that all intrahepatic cholangiocarcinoma should be transplanted, but because they showed how profoundly tumour burden and host context shape outcome.

The second route is more provocative and more clinically relevant: Liver-limited, unresectable intrahepatic cholangiocarcinoma with durable control on therapy. In the prospective series reported by Lunsford et al.[32] and subsequently updated by McMillan et al.,[33] selected patients treated with neoadjuvant systemic therapy and then transplanted achieved encouraging medium-term and 5-year survival. Early TESLA data now support the same principle, namely that biological stability over time is central and that transplantation only becomes credible once the tumour has already behaved well.[34]

Policy has moved, but carefully. Current OPTN guidance allows consideration of biopsy-proven, unresectable, solitary intrahepatic cholangiocarcinoma or mixed hepatocellular-cholangiocarcinoma no larger than 3 cm in cirrhotic patients, provided there has been locoregional or systemic treatment, at least 6 months of radiological stability, and no extrahepatic disease.[6] That conservatism is appropriate. It acknowledges that intrahepatic cholangiocarcinoma has earned a place in transplant oncology while also recognising that the evidence base remains small, centre-specific and vulnerable to selection bias.[33–35]

The practical conclusion is straightforward. Intrahepatic cholangiocarcinoma should no longer be dismissed as a blanket contraindication. Equally, it should not be normalised too quickly. At present, the strongest indications are very early cirrhotic intrahepatic cholangiocarcinoma and exceptionally selected liver-limited disease with durable response to therapy. Outside those circumstances, the burden of proof remains high.[30–35]

COLORECTAL LIVER METASTASES: THE METASTATIC FRONTIER

No development in contemporary liver transplantation has generated more interest, scepticism and ethical scrutiny than transplantation for colorectal liver metastases. For years the idea seemed closed: Recurrence appeared inevitable and the use of a deceased-donor graft for metastatic colorectal cancer difficult to justify. The Oslo group reopened the question by showing that inevitability of recurrence did not necessarily translate into futility of treatment.[13–15]

Hagness et al.[13] first reported 5-year overall survival of about 60% after transplantation for carefully selected non-resectable colorectal liver metastases. SECA-II tightened selection further and raised 5-year survival to 83%, with disease-free survival still modest but with a recurrence pattern that was often slow-growing, pulmonary and potentially amenable to local therapy.[15] This was an important conceptual shift. It suggested that the meaningful end point in colorectal liver metastases may not be recurrence-free survival alone, but prolonged overall survival with salvageable relapse.[13–16]

TransMet moved the field a step further by providing comparative evidence rather than single-centre optimism. In patients with permanently unresectable, liver-only colorectal liver metastases responding to chemotherapy, transplantation plus chemotherapy produced 5-year overall survival of 73%, compared with 9% for chemotherapy alone.[17] For transplant oncology, that trial matters enormously. It does not make colorectal liver metastases a routine indication, but it does make the old reflex dismissal intellectually untenable.[16,17]

The current challenge is ruthless selection. Contemporary United States guidance expects histologically proven colon or rectal adenocarcinoma, liver-only disease, a resected primary with negative margins, at least 12 months from diagnosis of liver metastases, response to first-line systemic therapy, no lesion larger than 10 cm before treatment, carcinoembryonic antigen no higher than 80 ng/mL, no extrahepatic disease on cross-sectional imaging and positron emission tomography, microsatellite-stable disease, and BRAF wild-type biology.[6] These are not bureaucratic hurdles. They are an attempt to translate outcome data into fair national practice.

The logic becomes clearer when the Oslo studies are read alongside the IHPBA consensus. High carcinoembryonic antigen levels, large lesions, progression on chemotherapy, a short interval from primary tumour treatment to transplantation, and unfavourable biology all indicate that the disease will outrun the graft.

Table 2 Practical selection variables and red flags in contemporary transplant-oncology assessment

Disease	Features that support listing	Red flags / likely contraindications	Practical message
Hepatocellular carcinoma	Falling or low alpha-fetoprotein; response to locoregional therapy; prolonged stability; tumour burden within accepted downstaging limits	Rising alpha-fetoprotein despite therapy; rapid progression; macrovascular invasion; extrahepatic spread; inability to downstage	Downstaging is a biological filter, not a technical gateway.
Perihilar cholangiocarcinoma	Clear protocol eligibility; completion of neoadjuvant pathway; negative staging for metastases; disease confined to the hilum	Transperitoneal biopsy; metastatic disease; protocol deviation; inability to complete staging or neoadjuvant treatment	In perihilar cholangiocarcinoma, the protocol itself is part of the treatment.
Intrahepatic cholangiocarcinoma	Solitary small tumour in cirrhosis or liver-limited disease with durable response; prolonged radiological stability; no nodal or extrahepatic disease	Multifocal or bulky disease; progression on systemic therapy; extrahepatic spread; biologically aggressive relapse pattern	Best considered within trial-like governance or prospective audit.
Colorectal liver metastases	Liver-only disease; resected primary; response to first-line chemotherapy; low carcinoembryonic antigen; favourable biology such as BRAF wild-type and microsatellite stability	Progression on chemotherapy; high carcinoembryonic antigen; large dominant lesion; short interval from primary treatment; extrahepatic disease; unfavourable molecular profile	The realistic goal is prolonged survival with manageable recurrence, not zero recurrence.

[15,16,18,19] Colorectal liver metastases are not an indication to be rescued by surgical bravado. It is an indication in which the operation makes sense only once the biology has already behaved well.

Living donor liver transplantation has become central in this space. Recent North American experience, including the Rochester programme, shows that highly structured living-donor pathways can provide transplantation to selected patients with colorectal liver metastases without directly competing for scarce deceased-donor grafts.[18,20,21] That does not abolish the ethical question; it changes its shape. The discussion is no longer only about justice between candidates on the waiting list, but also about the acceptability of exposing a healthy donor to risk for metastatic disease. Recurrence remains common and should be discussed plainly, but pulmonary relapse may be slow-growing and treatable. The realistic aim is not zero recurrence. It is a substantial prolongation of life, ideally with preserved function and access to salvage therapy.[15,16,18–22]

CROSS-CUTTING LESSONS

Across all four disease groups, the same principles recur. Morphology has become necessary but insufficient. Tumour size, number and distribution still matter, but they are now only the entry point. Alpha-fetoprotein trajectory in hepatocellular carcinoma, protocol completion in perihilar cholangiocarcinoma, radiological stability in intrahepatic cholangiocarcinoma, and molecular profile plus chemotherapy response in colorectal liver metastases all reflect the same deeper idea: Behaviour under treatment is a better proxy for transplantability than anatomy alone (Table 2).[4,5,10,16,19,21–23,34]

Pre-transplant therapy should therefore be understood as part of the selection process rather than as a delay before the 'real' treatment. Downstaging, chemoradiation and systemic therapy all function as biological filters. That is why time on treatment is valuable information rather than wasted time. The same is true after transplantation: Surveillance intensity, willingness to treat limited recurrence, and immunosuppression strategy all influence the real benefit obtained from a graft.[9,15–17,23,29,33]

Centre effect remains real. The best results in transplant oncology do not come from isolated technical excellence in the operating theatre. They come from integrated programmes in which radiology, interventional oncology, hepatology, medical oncology, pathology, anaesthesia, surgery and structured surveillance are aligned. This matters most for uncommon indications, where seemingly small lapses in staging or protocol discipline can completely alter outcome.[9,12,16,19,23,33]

Allocation ethics remain central. New indications should not be advanced simply because transplantation can technically be performed. They should advance when alternative therapies have been honestly compared, when denominator data are transparent, and when the magnitude of benefit is strong enough to justify the use of a scarce graft. That threshold will dif-

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fer among hepatocellular carcinoma, perihilar cholangiocarcinoma, intrahepatic cholangiocarcinoma, and colorectal liver metastases because the maturity of the evidence varies.[6,10,16,17,19,21–23]

FUTURE DIRECTIONS

The next phase of transplant oncology is unlikely to be driven by crude expansion of size-based criteria. The bigger gains will probably come from finer biological discrimination, including better integration of molecular profiling, radiomics, circulating biomarkers and response-adapted selection models.[5,11,12,21,23,34]

Just as important will be a better study design. Future reports need to describe referral volume, exclusions before listing, dropout during therapy, reasons for non-transplantation, recurrence pattern and salvage treatment, because post-transplant survival curves alone tell only part of the story.[16,17,19,21–23] The same priorities now recur across recent consensus statements, narrative reviews and programme reports: Dynamic biomarkers in hepatocellular carcinoma, protocol fidelity in cholangiocarcinoma, and denominator-based governance in colorectal liver metastases.[36–50]

CONCLUSIONS

Liver transplantation for cancer has moved well beyond a debate about whether malignancy should ever be an indication. The more useful question is which cancers, in which biological states, under which protocols, justify a graft. Hepatocellular carcinoma remains the benchmark and the most mature model of biological selection beyond anatomy. Perihilar cholangiocarcinoma is now an established indication when managed within a rigorous neoadjuvant and staging pathway. Intrahepatic cholangiocarcinoma has entered legitimate transplant practice, but only in carefully circumscribed settings. Colorectal liver metastases have crossed from bold experiment to serious clinical option in expert programmes, although they remain the most ethically and scientifically demanding indication.

The central lesson is shared across the whole field. Good transplant oncology does not come from stretching criteria until they break. It comes from using morphology, biology, treatment response and time to identify the uncommon patient whose cancer remains transplantable. The future of the field will depend less on how far criteria can be widened and more on how carefully candidates are chosen, and outcomes are audited.

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