Cureus
Part of SPRINGER NATURE

Open Access Review Article

Rethinking Endoscopic Surveillance in Primary Sclerosing Cholangitis With Inflammatory Bowel Disease: A Practical Review for Gastroenterologists and Endoscopists

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Review began 11/08/2026
Review ended 11/14/2026
Published 11/21/2026

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DOI: 10.7759/cureus.120066

Abstract

Primary sclerosing cholangitis (PSC) is the hepatobiliary complication of inflammatory bowel disease (IBD) that most directly reshapes endoscopic practice. It converts a routine colitis follow-up into a dual-organ cancer surveillance problem, and it places the gastroenterologist, rather than the hepatologist, at the decision point for both the colon and the bile duct. This review examines, from the perspective of the practising endoscopist, the evidence underpinning biliary and colorectal surveillance in PSC-IBD and proposes an integrated, resource-aware care pathway applicable outside high-volume Western transplant centres. A structured narrative review was performed. PubMed and Embase were searched from inception to July 2026 using controlled vocabulary and free-text terms for PSC, IBD, cholangiocarcinoma, cholangioscopy, biliary stricture, dysplasia, and surveillance. Clinical practice guidelines, randomised trials, meta-analyses, and large prospective cohorts were prioritised. Four practice-shaping themes emerged. First, the benefit of routine biliary imaging for cholangiocarcinoma surveillance remains uncertain, and its present value lies more in risk stratification and transplant triage than in curative early detection. Second, tissue acquisition during endoscopic retrograde cholangiopancreatography (ERCP) remains the weakest link in the pathway: a positive cholangioscopic biopsy is informative, but a negative result cannot exclude malignancy. Third, for a symptomatic biliary stricture, balloon dilatation rather than short-term stenting should be the initial endoscopic treatment. Fourth, colorectal surveillance is the intervention with the clearest benefit-to-risk ratio in PSC-IBD, yet it is the one most often delivered incompletely in routine practice. In PSC-IBD, the endoscopist should invest maximal effort in the colon, apply ERCP selectively and therapeutically rather than diagnostically, and recognise that biliary surveillance is currently a holding strategy pending better biomarkers. A pragmatic pathway suited to centres without on-site liver transplantation is proposed.

Categories: Oncology, Internal Medicine, Gastroenterology
Keywords: cholangiocarcinoma, cholangioscopy, chromoendoscopy, dysplasia surveillance, ercp, inflammatory bowel disease, primary sclerosing cholangitis

Introduction And Background

Primary sclerosing cholangitis (PSC) is a chronic, progressive, fibro-inflammatory cholangiopathy of unknown aetiology for which no medical therapy has been shown to alter the natural history. Its clinical importance to the gastroenterologist is disproportionate to its prevalence because approximately 60-80% of patients also have inflammatory bowel disease (IBD), and because the combination generates two independent malignancy risks, cholangiocarcinoma (CCA) and colorectal cancer (CRC), that must be managed simultaneously and largely endoscopically [1-3].

Extraintestinal manifestations affect roughly one in four patients with ulcerative colitis, and PSC is the most specific of the hepatobiliary complications [2,4]. The relationship is not symmetrical: whereas most patients with PSC have IBD, only approximately 2-8% of patients with IBD develop PSC [1,2]. PSC itself is heterogeneous: classic large-duct disease accounts for most cases and generates most endoscopic decisions, whereas small-duct PSC (cholestatic biochemistry and compatible histology with a normal cholangiogram) and PSC with features of autoimmune hepatitis are less common phenotypes with distinct prognoses [1,2]. When PSC and IBD coexist, the phenotype of the colitis changes, the surveillance interval changes, the risk of pouch complications after surgery changes, and the risk of disease recurrence after liver transplantation changes. PSC-IBD therefore behaves less like ulcerative colitis with a liver problem and more like a distinct clinical entity [3,5].

Three international guidelines, from the American Association for the Study of Liver Diseases (AASLD), the European Association for the Study of the Liver (EASL), and the British Society of Gastroenterology/UK-PSC group, now converge on a broadly similar surveillance framework [1-3]. What they do not resolve is the operational question that confronts the endoscopist: which patient should undergo endoscopic retrograde

How to cite this article
Al Shamousi K, Al Salmi U A, Al-Naamani Z R, et al. (November 21, 2026) Rethinking Endoscopic Surveillance in Primary Sclerosing Cholangitis With Inflammatory Bowel Disease: A Practical Review for Gastroenterologists and Endoscopists. Cureus 18(11): e120066. DOI 10.7759/cureus.120066

cholangiopancreatography (ERCP), when, with what tissue-acquisition strategy, and with what expectation of benefit. That gap matters because ERCP in PSC is not a low-risk procedure and because recent prospective data have called the effectiveness of biliary surveillance itself into question [6,7].

This review addresses PSC-IBD from the endoscopist's chair. It is deliberately organised around decisions rather than organ systems, and it closes with a pragmatic pathway designed for centres, including those across the Gulf and the wider Middle East, that manage these patients without on-site liver transplantation. Its explicit aim is therefore twofold: to appraise the evidence behind each endoscopic decision in PSC-IBD and to translate that evidence into a care pathway deliverable by gastroenterology services in the Gulf region and comparable settings, where liver transplantation requires external referral.

Review

Methods

PubMed/MEDLINE and Embase were searched from inception to July 31, 2026. Search strings combined MeSH terms and free-text terms for "cholangitis, sclerosing", "inflammatory bowel disease", "cholangiocarcinoma", "cholangioscopy", "biliary stricture", "colorectal neoplasms", "chromoendoscopy", and "surveillance". Reference lists of retrieved guidelines and systematic reviews were hand-searched. Priority was given to clinical practice guidelines, randomised controlled trials, systematic reviews with meta-analysis, and prospective multicentre cohorts; retrospective series were included where higher-level evidence was absent. Only English-language full-text publications were included. The review was prepared in accordance with the Scale for the Assessment of Narrative Review Articles (SANRA). In PubMed, the search logic was: ("Cholangitis, Sclerosing"[MeSH] OR "primary sclerosing cholangitis"[tiab]) AND ("Inflammatory Bowel Diseases"[MeSH] OR "inflammatory bowel disease"[tiab] OR "Cholangiocarcinoma"[MeSH] OR cholangioscopy[tiab] OR "biliary stricture"[tiab] OR "Colorectal Neoplasms"[MeSH] OR chromoendoscopy[tiab] OR surveillance[tiab]). Equivalent Emtree headings and free-text terms were used in Embase. Study selection was performed by the first author, and the final set of included studies was reviewed and agreed upon by all authors, with disagreements resolved through discussion. Each reference was verified by the first author against its digital object identifier and PubMed record before inclusion. Because this is a narrative rather than a systematic review, record-level screening counts were not formally tabulated, and no formal risk-of-bias assessment of individual studies was undertaken; these limitations are discussed at the end of the review.

Epidemiology and phenotype: why PSC-IBD is a separate disease

The colitis of PSC-IBD is characteristically extensive but quiescent. Pancolitis predominates, and rectal sparing and backwash ileitis are over-represented relative to ordinary ulcerative colitis; in a Southeast Asian paediatric cohort, 95% had pancolitis, 27% had rectal sparing, and 11% had backwash ileitis at presentation, and two-thirds achieved quiescent bowel disease on immunosuppression while the cholangiopathy continued to progress [8]. This dissociation, a bowel that looks well and a biliary tree that does not, is the single most important phenotypic point for the endoscopist because symptom burden is a poor guide to either colonic neoplastic risk or hepatic progression.

Paradoxically, the presence of IBD appears prognostically favourable for the liver. In the largest paediatric series assembled to date (781 children, 76% with IBD), event-free survival was 70% at five years and 53% at ten years, and both the PSC-IBD and small-duct phenotypes were independently associated with better outcomes [9]. Both of these series were paediatric, and their phenotypic and prognostic findings should be extrapolated to adults with caution. In adults, the Swedish SUPRIM (Swedish initiative for the study of Primary sclerosing cholangitis) cohort of 512 prospectively followed, unselected patients, the same cohort in which the prospective surveillance programme discussed below was evaluated [10], recorded liver transplantation in 10% and hepatobiliary malignancy in 3% over five years, underscoring both the heterogeneity of the disease and the low absolute event rate against which any surveillance strategy must be judged [11].

Making the diagnosis without harming the patient

The first and most consequential endoscopic decision in PSC is the decision not to perform ERCP. Contemporary guidance is explicit that magnetic resonance imaging with cholangiopancreatography (MRCP) is the diagnostic modality of choice and that ERCP should be avoided for the purpose of diagnosing PSC; where the initial study is inconclusive, the correct next step is a repeat high-quality MRCP rather than a diagnostic cholangiogram [1,2]. The justification is quantitative. A systematic review and meta-analysis comparing ERCP outcomes in patients with and without PSC found a threefold increase in the 30-day odds of post-procedural cholangitis (OR 3.26, 95% CI 1.08-9.90) despite antibiotic prophylaxis in the majority [6].

Equally important is terminology. AASLD guidance replaced the older, radiologically defined "dominant stricture" with the clinically anchored concept of a relevant stricture, any biliary or hepatic duct stricture accompanied by signs or symptoms of obstructive cholestasis and/or bacterial cholangitis [1]. This is not a semantic refinement. It converts an imaging finding into a clinical indication, and it removes the justification for intervening on a narrow duct in an asymptomatic patient with stable biochemistry. In

practice, the endoscopist should be able to answer, before scheduling ERCP, which of three questions the procedure is intended to settle: is there obstructive cholestasis or cholangitis requiring drainage; is there a suspicion of malignancy requiring tissue; or is there neither, in which case the procedure should not proceed.

Conversely, colonoscopy is underused at diagnosis. Guidance recommends ileocolonoscopy with biopsies in every patient with newly diagnosed PSC and no known IBD, repeated at approximately five-year intervals until IBD is identified, since histological colitis is frequently present in the absence of symptoms [1]. Registry data show this recommendation is honoured in only about four-fifths of eligible patients, even in well-resourced systems [12]. The guideline positions most relevant to endoscopic practice are summarised in Table *1*.

Question	Broad guideline position	Practical implication for the endoscopist
Diagnosis of PSC	MRCP first; ERCP not to be used diagnostically[1,2]	A diagnostic-only ERCP is now difficult to defend
Terminology	"Relevant stricture" = stricture with obstructive cholestasis and/or bacterial cholangitis [1]	Symptoms and biochemistry, not duct calibre, trigger intervention
Colonoscopy at PSC diagnosis	Ileocolonoscopy with biopsies in all; repeat ~5-yearly if negative[1]	Book the colonoscopy at the time of PSC diagnosis, not at first bowel symptom
CRC surveillance in PSC-IBD	Annual colonoscopy from IBD diagnosis [1,2,7,13]	No 8–10 year grace period; interval is annual from the outset
CCA surveillance	Annual imaging (MRI/MRCP or ultrasound); CA 19-9 optional per AASLD [1] but not routinely recommended per EASL [2]	Offer it, but counsel honestly about its limitations [10]
Tissue at suspected CCA	ERCP with brush cytology and FISH[1]	Add cholangioscopy with targeted biopsy where available [14]
UDCA	Not recommended at high dose; low-to-moderate dose optional [1,2]	Do not use biochemical response to justify escalating dose [15,16]

TABLE 1: Convergent and divergent guideline positions relevant to endoscopic practice

PSC: primary sclerosing cholangitis; MRCP: magnetic resonance cholangiopancreatography; ERCP: endoscopic retrograde cholangiopancreatography; CRC: colorectal cancer; IBD: inflammatory bowel disease; CCA: cholangiocarcinoma; MRI: magnetic resonance imaging; AASLD: American Association for the Study of Liver Diseases; EASL: European Association for the Study of the Liver; FISH: fluorescence in situ hybridisation; UDCA: ursodeoxycholic acid.

Biliary malignancy surveillance: an honest appraisal

The lifetime risk of CCA in PSC is most often quoted at 10-20%, and this risk underpins every guideline recommendation for annual hepatobiliary imaging [1,2,7]. The difficulty is that recommending surveillance and demonstrating that surveillance works are different propositions.

The most rigorous test to date is the Swedish nationwide prospective study of 512 unselected patients with PSC (the SUPRIM cohort introduced above [11]) who were followed with annual contrast-enhanced MRI/MRCP and CA 19-9, with ERCP triggered by severe or progressive bile duct changes [10]. Over five years, 11 patients (2%) were diagnosed with CCA and two (0.5%) with high-grade biliary dysplasia. Severe or progressive duct changes on MRI/MRCP were strongly associated with the composite of high-grade dysplasia or hepatobiliary malignancy (HR 10.50, 95% CI 2.49-44.31; an imprecise estimate based on only 13 events), as were rising CA 19-9 levels. Yet the median survival of patients diagnosed with CCA was only 13 months. The authors' conclusion was unambiguous: this strategy did not detect cancer early enough to support long-term survival.

Guidelines also diverge on the role of CA 19-9. AASLD guidance permits annual cross-sectional imaging with or without CA 19-9 [1], whereas EASL does not recommend its routine use for surveillance, reflecting its limited specificity and frequent elevation during bacterial cholangitis or biliary obstruction [2]. The 2025 EASL guideline on extrahepatic cholangiocarcinoma has since updated recommendations on the classification and diagnostic work-up of perihilar and distal CCA [17]. In practice, CA 19-9 is best interpreted as a trend alongside imaging rather than as a stand-alone surveillance test.

Two inferences follow, and they are not contradictory. The first is that annual imaging retains value as a risk-stratification and transplant-triage tool: progressive duct change is a genuine warning signal, and a

small number of patients are transplanted for biliary dysplasia rather than for end-stage liver disease [10]. The second is that clinicians should stop describing biliary surveillance to patients as an intervention that will find cancer in time. It is, at present, a holding strategy. Framing it accurately protects both the patient's autonomy and the clinician's credibility when a cancer is nonetheless diagnosed at an incurable stage.

If imaging is primarily a risk-stratification tool, it should be interpreted alongside validated prognostic instruments. Clinical scores such as the UK-PSC risk score [18] and the Amsterdam-Oxford model [19], non-invasive fibrosis assessment with the enhanced liver fibrosis test or elastography, and MRCP-based scoring systems are recognised in current guidance as aids to prognostication [1,2], and they offer a more coherent basis for setting surveillance intensity and the timing of transplant referral than imaging appearance alone.

Real-world performance compounds the problem. In a 1,300-patient North American registry analysis spanning 19 centres, adherence to abdominal imaging for hepatobiliary cancer surveillance fell from 74% in year one to 51% by year four, CA 19-9 was documented in only 47% over five years, and annual colonoscopy in PSC-IBD was performed in just 30-35% of patients [12]. Lower education and household income independently predicted non-adherence. A surveillance programme that is neither effective in trials nor delivered in practice requires redesign, not repetition.

Tissue acquisition: the weakest link in the pathway

When a relevant stricture requires evaluation, the endoscopist must choose among brush cytology, fluorescence in situ hybridisation (FISH), cholangioscopy with targeted biopsy, and probe-based confocal laser endomicroscopy (pCLE). The comparative evidence is summarised in Table *2* and is more sobering than commonly assumed.

Modality	Pooled/reported sensitivity	Pooled/reported specificity	Practical comment
Brush cytology	43% (pooled) [20]; 47% (single study) [21]	97% (pooled) [20]; 95% (single study) [21]	Widely available; low sensitivity is the accepted trade-off
Brushing + FISH	Incremental over cytology alone [14]	High [14]	Guideline-endorsed combination for suspected CCA [1]
DSOC, visual impression	64% (single study) [21] to 65% (pooled) [14]	62% (single study) [21] to 97% (pooled) [14]	Wide variation; PSC mucosa is diffusely abnormal
DSOC, targeted biopsy	15% (single study) [21] to 65% (pooled) [14]	97% [14]	A positive result is near-definitive; a negative one is not
DSOC + narrow-band imaging	No improvement over white light [22]	—	48% more biopsies, no gain in dysplasia detection
pCLE	AUC 0.72 (single study) [23]	—	Not superior to direct visualisation in comparative data
AI-assisted DSOC	AUC 0.79 (single study) [23]	—	Promising; requires prospective head-to-head validation

TABLE 2: Performance of ERCP-based tissue-acquisition modalities in PSC

Values are drawn from heterogeneous populations and are not directly comparable across rows. Pooled values derive from meta-analyses; values labelled single study derive from individual cohorts. The single-centre cholangioscopy series included patients with indeterminate strictures of mixed aetiology, of whom 40% had PSC.

FISH: fluorescence in situ hybridisation; CCA: cholangiocarcinoma; DSOC: digital single-operator cholangioscopy; PSC: primary sclerosing cholangitis; pCLE: probe-based confocal laser endomicroscopy; AUC: area under the receiver operating characteristic curve; AI: artificial intelligence; ERCP: endoscopic retrograde cholangiopancreatography.

A meta-analysis of 21 studies evaluating ERCP-based modalities in PSC found single-operator cholangioscopy with targeted biopsy to be the most accurate approach overall (96%, 95% CI 94-97), with a pooled sensitivity of 65% (95% CI 35-87) and specificity of 97% (95% CI 87-99) [14]. The confidence interval around the sensitivity estimate is wide, reflecting small and heterogeneous studies. High specificity is genuinely useful: a positive cholangioscopic biopsy is close to definitive. But sensitivity in the region of two-thirds means that a negative result cannot exclude malignancy, and this asymmetry must govern how the result is communicated to the multidisciplinary team.

For brush cytology alone, a separate meta-analysis of 11 studies including 747 patients with PSC reported a

pooled sensitivity of 43% and specificity of 97% for CCA [20]. Single-centre real-world data are less flattering still. In a series of 80 patients undergoing single-operator peroral cholangioscopy for indeterminate strictures, 40% of whom had PSC, visual impression achieved a sensitivity of 64% and specificity of 62%, and targeted biopsies achieved a sensitivity of only 15%; sequential brush cytology performed better (sensitivity 47%, specificity 95%), and cholangioscopy altered management in just 17% of patients [21]. The discrepancy with the pooled estimates is instructive: prior stent placement, a background of diffuse PSC-related mucosal irregularity, and operator experience all degrade performance. In PSC specifically, the entire biliary mucosa may appear abnormal, and the visual criteria validated in non-PSC indeterminate strictures do not transfer cleanly.

Advanced imaging has so far failed to close the gap. Cholangioscopy with narrow-band imaging in a prospective PSC cohort increased the number of suspicious lesions biopsied by 48% without improving the dysplasia detection rate, although it did permit delineation of tumour margins in extrahepatic CCA [22]. Artificial intelligence is an emerging but not yet decisive adjunct: a retrospective comparison of a cholangioscopy-based neural network against pCLE reported an area under the curve of 0.79 for the artificial intelligence model versus 0.72 for pCLE, 0.74 for direct visualisation, and 0.83 for guided biopsy, with no statistically significant separation between them [23]. The honest reading is that no currently available modality reliably rules out malignancy in a PSC stricture, and that serial assessment over time frequently substitutes for a single definitive test.

Molecular adjuncts are the most promising route to closing this gap. Adding a targeted next-generation sequencing panel (BiliSeq) to ERCP-obtained biliary specimens increased sensitivity for malignant strictures while maintaining specificity, and in the subgroup with PSC, its sensitivity was 83% compared with 8% for pathological evaluation [24]. These data come from a single referral programme and require prospective validation in PSC-specific cohorts, but they suggest that the next gain in tissue diagnosis is more likely to come from molecular analysis than from further refinement of visual inspection.

Therapeutic ERCP: one question that randomised evidence has settled

Amid considerable uncertainty, the management of the symptomatic stricture is unusually well defined. In a multicentre randomised trial conducted across nine European tertiary centres, patients with PSC and a dominant stricture were allocated to balloon dilatation or short-term (maximum two weeks) stenting [25]. Recruitment was terminated early for futility and for a difference in harm. Cumulative recurrence-free patency at 24 months did not differ (0.34 for stenting versus 0.30 for dilatation), while treatment-related serious adverse events occurred in 45% of the stent group compared with 6.7% of the dilatation group (OR 11.7, 95% CI 2.4-57.2). The confidence interval is wide, reflecting early termination, but the direction of harm is consistent. Balloon dilatation is therefore the initial treatment of choice, a point of particular relevance for patients with an intact papilla.

Periprocedural risk mitigation is less settled. Beyond the elevated odds of cholangitis already noted, a meta-analysis identified inadvertent guidewire passage into the pancreatic duct (OR 7.44, 95% CI 3.33-16.65) and biliary sphincterotomy (OR 4.80, 95% CI 1.92-12.03) as the dominant modifiable risk factors for post-ERCP pancreatitis in this population [6]. Both are technique-dependent and argue for wire-guided cannulation by an experienced operator and for restraint in performing sphincterotomy where it is not needed for the therapeutic objective.

Notably, the standard pharmacological prophylaxis may not transfer to PSC. In an observational case-control analysis of 2,000 ERCPs performed in 931 patients with PSC, rectal diclofenac did not significantly reduce post-ERCP pancreatitis (4.9% versus 6.2%), even though the rate among patients with a native papilla remained clinically meaningful at around 9-11% [26]. This should not be read as a licence to omit rectal non-steroidal prophylaxis, which remains inexpensive, low-risk, and supported by broader evidence; it should be read as a caution that PSC cohorts differ from the mixed populations in which prophylaxis was validated, and as a call for a targeted randomised trial in high-risk PSC procedures.

Colorectal neoplasia: where endoscopy still earns its place

If biliary surveillance in PSC-IBD is currently a holding strategy, colorectal surveillance is the opposite. The magnitude of risk is not in dispute. PSC-IBD carries an approximately tenfold increase in CRC risk relative to the general population [7], and a meta-analysis of 11 studies found an approximately fivefold higher risk of colorectal neoplasia in ulcerative colitis with PSC than in ulcerative colitis alone (OR 4.79, 95% CI 3.58-6.41) [27]. In a tertiary-centre cohort, patients with PSC-IBD were far more likely than those with PSC alone to be diagnosed with CRC (23.3% versus 1.8%, $P < 0.01$) or with dysplasia (16.7% versus 0%, $P < 0.01$); notably, patients with PSC alone still had a significantly elevated standardised incidence ratio of 9.2 (95% CI 1.1-33.2) compared with the background population [28]. As a tertiary-centre comparison, these figures are susceptible to referral bias, and the wide confidence interval around the standardised incidence ratio for PSC alone indicates considerable imprecision. For context, a recent review estimates the 20-year CRC risk in ulcerative colitis without PSC at approximately 4.5%, a 1.7-fold increase over the general population [4].

Two operational consequences follow. First, surveillance in PSC-IBD begins at the time of IBD diagnosis and

is annual; there is no eight-to-ten-year grace period [1,2,7]. Second, because dysplasia in this group is disproportionately flat and right-sided, technique matters more than in ordinary colitis surveillance.

On technique, the field had been drifting towards virtual chromoendoscopy on grounds of convenience. A recent prospective randomised study of 300 patients from Kuwait, performed on a single imaging platform, provides a useful corrective: methylene blue dye-based chromoendoscopy achieved a higher per-patient dysplasia detection rate than blue light imaging and linked colour imaging (12% versus 8%, P = 0.04), with the advantage concentrated in flat and proximal lesions, and PSC emerged on multivariable analysis as an independent predictor of dysplasia detection alongside disease duration of ten years or more and pancolitis [29]. The authors concluded that dye-based chromoendoscopy should remain the preferred technique in high-risk populations, reserving virtual chromoendoscopy for lower-risk settings. That conclusion maps directly onto PSC-IBD, and it is drawn from a regional population with a case mix comparable to that of Oman and the wider Gulf.

This conclusion should nonetheless be weighed against the wider evidence. The SCENIC (Surveillance for Colorectal Endoscopic Neoplasia Detection and Management in Inflammatory Bowel Disease Patients: International Consensus Recommendations) consensus recommended dye-based chromoendoscopy and did not endorse narrow-band imaging in place of white light [30], yet a meta-analysis of head-to-head comparisons, albeit based on only two studies, found no significant difference in neoplasia detection between dye-based chromoendoscopy and narrow-band imaging [31]. The 2025 British Society of Gastroenterology (BSG) guideline, informed by a network meta-analysis of 26 randomised trials, suggests dye-based chromoendoscopy for its small benefit over high-definition white light and was unable to make a recommendation on virtual chromoendoscopy because of very low-certainty evidence [13]. Our preference for dye spraying in PSC-IBD therefore reflects a judgment for the highest-risk group rather than a settled conclusion. The same guideline advises that additional quadrantic non-targeted biopsies every 10 cm, or from each colonic segment, be considered in patients with PSC even when dye spray is used, alongside targeted biopsies of visible lesions [13].

High-definition white light with targeted biopsies remains an acceptable alternative where dye spraying is impractical, and the trajectory of the field is towards precision rather than random sampling, with stool-based markers and artificial intelligence under active evaluation [32,33]. Decisions about when to stop surveillance should be individualised through shared decision-making, taking account of chronological age, comorbidity, life expectancy, and prior surveillance findings rather than a fixed cut-off [34].

Treating the bowel without treating the liver

No drug has been shown to modify the natural history of PSC, although candidate agents remain under evaluation. Norursodeoxycholic acid, for example, reduced alkaline phosphatase in a phase 2 randomised trial [35], and the endoscopist should be familiar with the two most consequential negative results. High-dose ursodeoxycholic acid (28-30 mg/kg/day) improved serum liver tests but was associated with a 2.3-fold increase in the risk of reaching a clinical endpoint and a higher rate of serious adverse events (63% versus 37%) in a randomised, double-blind, placebo-controlled trial terminated for futility [15]. Mechanistic work from the same cohort demonstrated marked expansion of the total bile acid pool, including the hepatotoxic secondary bile acid lithocholic acid [16]. The practical lesson is that biochemical improvement in PSC is not a valid surrogate for benefit, a principle that also constrains the interpretation of alkaline phosphatase responses to IBD therapy.

Advanced therapies for the bowel disease behave as one would expect for the bowel and disappointingly for the liver. In a multicentre retrospective study of 34 patients with PSC-IBD treated with vedolizumab, there was no change in alkaline phosphatase at week 30 despite a plausible mechanistic rationale for gut-liver lymphocyte trafficking blockade, while clinical remission was achieved in 55% of patients with Crohn's disease and 29% of patients with ulcerative colitis, with a favourable safety profile [36]. A broader appraisal of anti-tumour necrosis factor agents, vedolizumab, interleukin-12/23 inhibitors, Janus kinase inhibitors, and thiopurines in PSC-IBD concluded that no agent has yet reduced disease-related morbidity or mortality, that the evidence base is dominated by small retrospective series with heterogeneous outcomes, and that the absence of a validated surrogate endpoint in PSC is itself a barrier to progress [37].

The clinical corollary is straightforward and worth stating plainly to patients: choose the IBD therapy on the merits of the colitis, monitor the liver independently, and do not expect one to rescue the other.

The pouch, the graft, and the sequence in which they are considered

Colectomy with ileal pouch-anal anastomosis (IPAA) is common in PSC-IBD, and pouchitis is substantially more frequent than in ulcerative colitis without PSC. In a large institutional pouch registry of 160 patients with PSC-IBD, chronic pouchitis affected 67% of transplanted patients compared with 45.5% of non-transplanted patients, although overall and pouch survival were preserved and PSC recurrence was 6% at ten years [38]. A meta-analysis of seven studies including 291 patients with PSC, IBD, and liver transplantation found a pooled pouchitis risk of 65% and, importantly for surgical sequencing, a significantly higher risk when IPAA preceded transplantation (83%) than when transplantation preceded IPAA (59%, P < 0.001) [39].

These data do not establish causation, but they justify explicit multidisciplinary discussion of sequencing in the patient who is likely to need both operations.

Recurrent PSC (rPSC) after transplantation affects 15-35% of recipients at five to ten years, with rates increasing over time and no effective medical therapy available [40]. A systematic review and meta-analysis of 2,159 patients identified rPSC in 17.7% and quantified several risk factors: colectomy before transplantation was protective (HR 0.65, 95% CI 0.42-0.99), whereas retained IBD (HR 1.73), pre-transplant CCA (HR 2.42), donor age (HR 1.24 per decade), and acute cellular rejection (HR 1.94) increased risk [41]. Single-centre national programme data confirm that recurrence is common (20.7%) and occurs earlier and more frequently after re-transplantation [42]. The unifying theme, that control of intestinal inflammation is a potentially modifiable determinant of graft outcome, places the gastroenterologist, not only the transplant hepatologist, in the causal pathway [40].

A proposed pathway for centres without on-site transplantation

Regional data deserve emphasis. IBD incidence across the Arab world is rising from a historically low base: a systematic review and meta-analysis of 16 studies from Saudi Arabia, Egypt, Kuwait, the United Arab Emirates, Bahrain, Lebanon, and Oman produced pooled incidence rates of 2.33 per 100,000 person-years for ulcerative colitis and 1.46 per 100,000 person-years for Crohn's disease [43]. As prevalent cohorts age and disease duration lengthens, the regional PSC-IBD population, and with it the demand for advanced biliary endoscopy, will grow in centres that have not historically needed it. Building this capability is therefore a service-planning question, not merely a clinical one.

Table *3* sets out an integrated pathway, proposed by the authors rather than drawn from any single guideline, that reflects the evidence reviewed above and the constraints of a typical tertiary centre in the Gulf region: reliable access to MRCP and colonoscopy, ERCP available but not high-volume for PSC, cholangioscopy available at a single referral site, and liver transplantation requiring external referral. The organising principle is to concentrate endoscopic effort where the yield is demonstrable, to defer ERCP until a clinical trigger exists, and to define referral thresholds in advance rather than at the point of crisis.

Time point	Action	Rationale/evidence
At PSC diagnosis	Ileocolonoscopy with segmental biopsies even if asymptomatic; MRCP; baseline non-invasive fibrosis staging	Histological colitis is often silent; ERCP is not a diagnostic test [1,2,12]
At IBD diagnosis in known PSC	Begin annual surveillance colonoscopy immediately	Tenfold CRC risk; no 8-10 year grace period[7,28]
Annual	Colonoscopy with dye-based chromoendoscopy and targeted biopsies; consider additional quadrantic non-targeted biopsies every 10 cm or per segment; document withdrawal time and preparation quality	Higher detection of flat, right-sided dysplasia than virtual chromoendoscopy in a randomised study [29]; non-targeted biopsies per BSG 2025[13]
Annual	Contrast-enhanced MRI/MRCP; CA 19-9 optional as guidelines diverge; explicit counselling on limitations	Risk stratification and transplant triage rather than curative early detection [1,2,10]
Trigger for ERCP	Obstructive cholestasis, bacterial cholangitis, or progressive duct change on MRCP	The "relevant stricture" concept [1,10]
At ERCP	Balloon dilatation first; avoid routine short-term stenting; brush cytology + FISH; cholangioscopy with targeted biopsy where available; wire-guided cannulation; avoid unnecessary sphincterotomy	Short-term stenting: serious adverse events 45% vs 6.7% with dilatation, without improved patency [25]; wire passage and sphincterotomy drive PEP [6]
If surgery anticipated	Multidisciplinary discussion of colectomy/IPAA timing relative to possible transplantation	Pouchitis risk 83% when IPAA precedes transplant vs 59% when it follows [39]
Referral threshold	Any high-grade biliary dysplasia, unresectable hilar mass, decompensation, or recurrent cholangitis → transplant centre	Recurrent cholangitis attracts MELD exception points under US allocation policy [1]; Gulf programmes should apply local or referral-centre criteria

TABLE 3: Authors' proposed integrated care pathway in PSC-IBD

Original table created by the authors; the pathway is proposed by the authors, and the supporting evidence for each step is cited within the table. Steps supported by guideline documents reflect guideline recommendations; the sequencing and referral thresholds are the authors' proposal and have not been validated prospectively.

PSC: primary sclerosing cholangitis; MRCP: magnetic resonance cholangiopancreatography; IBD: inflammatory bowel disease; CRC: colorectal cancer; MRI: magnetic resonance imaging; ERCP: endoscopic retrograde cholangiopancreatography; FISH: fluorescence in situ hybridisation; BSG: British Society of Gastroenterology; PEP: post-ERCP pancreatitis; IPAA: ileal pouch–anal anastomosis; MELD: Model for End-Stage Liver Disease; US: United States.

Research priorities

Four questions are both unanswered and answerable. First, can a composite of serial MRCP changes, CA 19-9 trajectory, and circulating tumour DNA outperform annual imaging alone in identifying the small subgroup who benefit from intervention? The prospective Swedish experience suggests that improved diagnostics, not more frequent application of existing ones, is the necessary step [10]. Second, does rectal non-steroidal prophylaxis reduce post-ERCP pancreatitis specifically in PSC patients with a native papilla, a group in whom the event rate approaches 10% and in whom existing observational data are null [26]? Third, can artificial-intelligence-assisted cholangioscopy be validated prospectively against a balanced neoplastic and non-neoplastic case mix, rather than in cohorts where malignancy prevalence exceeds 95% [23]? Fourth, what is the true prevalence, phenotype, and outcome of PSC-IBD in Middle Eastern populations, where IBD incidence is rising and where no dedicated cohort currently exists [43]? A regional registry would be an achievable and high-value contribution.

Limitations

This is a narrative review and shares the limitations of that design. Study selection did not follow a preregistered systematic protocol, record-level screening counts were not tabulated, and no formal risk-of-bias assessment of individual studies was undertaken, so selection bias cannot be excluded. The search was restricted to English-language publications. Several sections, particularly those on tissue acquisition and pharmacological prophylaxis, rely on retrospective, single-centre, or observational data, and some phenotypic and prognostic data are drawn from paediatric cohorts. Estimates from heterogeneous populations, including mixed-aetiology stricture cohorts, are not directly comparable, and the proposed pathway represents the authors' synthesis rather than a prospectively validated algorithm.

Key messages

MRCP, not ERCP, is the diagnostic test for PSC. ERCP is reserved for a clinically relevant stricture or a need for tissue sampling. Annual colonoscopic surveillance should begin at IBD diagnosis in PSC-IBD. Dye-based chromoendoscopy with targeted biopsies is preferred, and additional quadrantic non-targeted biopsies should be considered. Annual biliary imaging is a risk-stratification and transplant-triage strategy rather than proven curative early detection, and patients should be counselled accordingly. Guidelines diverge on CA 19-9. No current tissue-acquisition modality reliably excludes malignancy in a PSC stricture. A positive cholangioscopic biopsy is informative, but a negative one is not. For a symptomatic dominant stricture, balloon dilatation is preferred to short-term stenting. Centres without on-site liver transplantation should define referral thresholds in advance.

Conclusions

PSC-IBD requires the endoscopist to manage two cancers with one scope and imperfect evidence. The defensible position in 2026 is one of asymmetric effort: maximal, technically rigorous annual colonoscopic surveillance, because the risk is large and the intervention demonstrably detects dysplasia; disciplined restraint in the biliary tree, with ERCP reserved for a clinically relevant stricture and balloon dilatation preferred to stenting; and candour with patients about the current limitations of cholangiocarcinoma surveillance. Adherence to the elements that work is poor even in well-resourced systems, which suggests that the most immediate gain available to most centres is not a new technology but the reliable delivery of what guidelines already recommend.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

Acknowledgements

Data sharing is not applicable to this article as no new data were created or analysed. A large language model (Claude, Anthropic PBC, San Francisco, California) was used to assist with literature retrieval, structuring and language editing of this manuscript. All content was reviewed, verified against primary sources and edited by the authors, who take full responsibility for the accuracy and integrity of the work. Every reference cited was independently verified against its digital object identifier and PubMed record.

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