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# Incisional hernia repair after abdominal organ transplantation: current evidence, controversies and research gaps

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Incisional hernia is a common late complication of abdominal organ transplantation, with reported frequency ranging from a pooled 4% after kidney transplantation to a prevalence of 43% on clinical examination after liver transplantation. Immunosuppression places recipients at least in Ventral Hernia Working Group grade 2, and concern about wound failure, mesh infection and recurrence often delays repair. This mini review synthesises evidence on elective repair in adult liver, kidney and pancreas recipients around three questions: whether transplant status independently predicts adverse outcome, how immunosuppression relates to outcome, and how operative access, reconstruction, mesh plane, material and fascial closure should be chosen. In most registry and matched comparisons, 30-day wound morbidity and length of stay were similar to those of non-transplant patients, although one matched laparoscopic series found more surgical site occurrences. Study-specific recurrence ranges from 0% in selected specialist series to 37%, with differing follow-up and ascertainment, and observational comparisons do not establish whether transplantation independently increases recurrence. A randomised trial showed more wound complications with *de novo* sirolimus after kidney transplantation; the association between maintenance sirolimus and recurrence after later repair rests on small, conflicting series, and perioperative conversion is untested. Recent series of transversus abdominis release report recurrence of 0%–25%, depending on incision, defect size, follow-up and comparator. Diaphragmatic tack fixation should be avoided. Two randomised trials of prophylactic mesh at transplant closure are registered, without published results. Research priorities include transplant-specific registry fields, standardised recurrence ascertainment, longer post-repair follow-up and graft-related outcomes.

KEYWORDS
**abdominal wall reconstruction, immunosuppression, incisional hernia, sirolimus, solid organ transplantation**

## Introduction

Incisional hernia (IH) is a frequent late complication of abdominal organ transplantation, and its reported frequency depends on the measure used. In 3,460 transplants at one institution, Kaplan–Meier cumulative incidence at 10 years was 7.0% after kidney, 19.0% after liver and 21.8% after pancreas transplantation [1]. After liver transplantation, cumulative incidence was 28.5% at 5 years [2] and 35.1% at a median of

76 months [3], whereas a cross-sectional study that examined every patient found a prevalence of 43% [4]. After kidney transplantation, the pooled proportion was 4% (95% CI 3%–5%) across 20 studies [5]. Variation reflects hernia definition, detection method and follow-up.

IH impairs health-related quality of life after liver transplantation [4], yet repair is approached with caution. In the Ventral Hernia Working Group (VHWG) system, immunosuppression places a recipient with a clean wound at least in grade 2, and previous wound infection or contamination raises the grade [6]. Reported recurrence after repair ranges from 0% to 76.9% [7]. Better survival after repair than without it [8] is open to selection and immortal-time bias, both expected to favour the repaired group.

Earlier reviews summarised incidence and outcomes by organ [5, 7, 9, 10]. Since then, a recurrence-focused liver cohort [3], series of transversus abdominis release (TAR) [11–13], a matched laparoscopic comparison [14] and two registered prophylactic mesh trials [15, 16] have been reported. This mini review integrates these data around three questions on which practice varies: whether transplant status independently predicts adverse outcome after repair, how immunosuppression relates to outcome, and how access, reconstruction, mesh and fascial closure should be chosen. Throughout, we separate direct evidence in liver, kidney and pancreas recipients from indirect evidence in mixed solid organ transplant (SOT) or non-transplant immunosuppressed cohorts.

## Literature identification and scope

We searched PubMed/MEDLINE, Embase, Scopus and the Cochrane Library from January 2000 to 30 September 2026, and ClinicalTrials.gov, combining terms for incisional or ventral hernia and repair with liver, kidney, pancreas or solid organ transplantation, immunosuppression, sirolimus or mammalian target of rapamycin (mTOR) inhibitors, and prophylactic mesh, and screened reference lists. We included English-language peer-reviewed studies of adult abdominal organ recipients reporting IH incidence, risk factors, repair outcomes or prevention, prioritising randomised trials, systematic reviews, comparative cohorts and series of at least 20 recipients. Case reports, video vignettes, conference abstracts, paediatric series and studies without separable transplant data were excluded. Non-transplant studies are cited only where transplant data were absent and are identified as indirect. Unless stated, evidence concerns elective repair and should not be extended to emergency repair, active infection, contaminated fields or unstable graft function.

## Risk factors for hernia formation

Surgical site infection (SSI) after transplantation is the most consistent predictor of later IH, with hazard ratios (HR) of 24.7 after kidney, 12.0 after liver and 12.95 after pancreas transplantation [1]. Obesity is the main modifiable factor: BMI >30 kg/m$^2$ (HR 2.9), female sex, smoking and repeated iliac fossa exploration predicted IH in 1,564 kidney recipients [17], and BMI ≥30 kg/m$^2$ (HR 2.67; 95% CI 1.7–4.3) and seroma predicted IH after liver transplantation, whereas induction agent did not [2]. Age, BMI and relaparotomy were independent predictors in a German liver centre [18]. Incision also matters: J-shaped incisions carried a lower IH rate than chevron or reverse-T incisions after liver transplantation [19], and Gibson a lower rate than hockey-stick incisions after kidney transplantation (4% vs. 16%) [20]. Cephalad extension of the Mercedes incision produces subxiphoid defects that are difficult to repair [21]. Whether these factors predict hernia size or time to repair, a different outcome, was examined in 31 recipients, too few to exclude a moderate effect [22].

## Is transplant status an independent risk factor?

Short-term comparisons are broadly reassuring but not uniform. In the Abdominal Core Health Quality Collaborative (ACHQC), 165 SOT recipients undergoing elective repair had an adjusted median length of stay similar to that of 13,452 non-transplant patients (2.8 vs. 2.6 days; p = 0.5), with similar SSI (2.4% vs. 4.0%; p = 0.42) and surgical site occurrence requiring procedural intervention (4.2% vs. 5.8%; p = 0.38) [23]. Prior liver or kidney transplantation was not associated with in-hospital mortality or stay after adjustment in the US National Inpatient Sample [24], and wound complications were similar after liver transplantation and hepatopancreaticobiliary surgery through the same incision [25]. In contrast, after propensity matching, liver recipients undergoing laparoscopic repair had more surgical site occurrences (SSO) than controls (19.0% vs. 2.4%; p = 0.029) [14]; defect size was not matched, so the excess may reflect hernia complexity rather than transplantation.

Recurrence estimates are study-specific and not interchangeable (Table 1). In a case-matched cohort of mixed SOT recipients (31 liver, 11 kidney, 3 heart, 2 islet), recurrence was 12.1% vs. 3.0% at a mean of 13 months (p = 0.16) [32]. These percentages correspond to 4/33 and 1/33, although the report describes 32 matched pairs, a discrepancy in the source. The crude risk ratio of 4.0 (95% CI approximately 0.5–34) is compatible with no difference or a large excess. In an Americas Hernia Society Quality Collaborative (AHSQC) analysis of 166 abdominal transplant recipients, composite 2-year recurrence was 28%, but only 75 patients (45%) contributed and the authors note low follow-up [29]; if follow-up depended on symptoms, the estimate could be biased in either direction. After a first open repair in 141 liver recipients, recurrence occurred in 50 (35.4%), and suture repair was associated with recurrence compared with mesh (subdistribution HR 2.48; 95% CI 1.25–4.90) [3]. Pooled recurrence in kidney recipients was 16% (95% CI 9%–23%) [5], whereas ACHQC recurrence of 0.6% [23] almost certainly reflects under-ascertainment.

Systemic morbidity is a separate concern. In 172 liver recipients, acute kidney injury (AKI), defined as a creatinine rise >0.3 mg/dL within 24 h, occurred in 48% after repair and was associated with longer stay and lower intraoperative fluid administration (6.0 vs. 8.5 mL/kg/h; p = 0.031) [34]; this incidence is specific to that cohort and definition.

Recipients offered elective repair are selected for fitness and favourable anatomy, but comparators are also selected, so the net

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TABLE 1 Selected studies reporting outcomes of incisional hernia repair in abdominal organ transplant recipients, ordered by year. Recurrence is given as reported, with numerator and denominator where available, follow-up (FU) from repair unless stated, and method of ascertainment. NR indicates not reported in the source.

| Study | Design and setting | Population | Repair (access; technique; mesh plane and material; closure) | Recurrence (n/N; follow-up; ascertainment) | Other principal findings | Main limitation |
|---|---|---|---|---|---|---|
| Andreoni [21] | Retrospective; single centre; USA | 27 immunosuppressed, mostly LT (mixed) | Lap IPOM (12 completed of 13 attempted) vs. open [15]; diaphragmatic tack fixation described* | Lap 0/12; open 7/15 with recurrence and/or serious mesh infection (composite); FU: NR; ascertainment: NR | Lower blood loss with lap | Unmatched; mixed population; composite outcome; small |
| Fikatas [18] | Single centre; described as prospective; Germany | 77 LT | Mixed mesh techniques | 12/77 (15.6%); IPOM 7.7% vs. other mesh 21.4%; median 7.9 months to recurrence; FU: NR; ascertainment: NR | Age, BMI and relaparotomy predicted IH | Technique not allocated; unadjusted |
| Weiss [26] | Retrospective; single centre; Austria | 54 LT | Lap IPOM | 9/54 (17%); FU: NR; ascertainment: NR | BMI ($p = 0.001$) and sirolimus ($p = 0.014$) associated with recurrence | Small sirolimus group; confounding by indication |
| Lam [25] | Retrospective; single centre; Belgium | 55 LT vs. 27 HPB (same incision) | Mostly synthetic mesh | LT 9/55 (16.4%) vs. HPB 3/27 (11.1%); FU: NR; ascertainment: NR | Similar wound complications; mTOR inhibitor associated with SSI; steroids, immunosuppression and non-synthetic mesh associated with recurrence | Small comparator; mixed techniques |
| Kuo [27] | Single centre; historical controls; Taiwan | 35 LT | Lap [18] vs. open [17] | Lap 0/18 to end of study period; open: NR; FU: NR (lap repairs March 2015 to March 2016); ascertainment: NR | Minor complications 16.7% vs. 52.9%; LOS 3 vs. 7 days | Historical controls; short FU |
| Tastaldi [11] | Two centres; AHSQC data; USA | 44 LT | Open TAR via midline incision; retromuscular synthetic mesh; fascial closure 41/44 | 11/44 (25%); median FU 13 months (minimum 12); ascertainment: NR | 8 recurrences from central mesh fracture; median width 20 cm | Large defects; no comparator; short FU |
| Frountzas [10] | Systematic review; proportional meta-analysis of primary cohorts | 18 studies; 640 LT with IH; 546 mesh repairs | Open vs. lap mesh repair | Not used: pooled outcome was postoperative complications; recurrence taken from primary studies in this table | Complications 144/546 (26%); pooled open 23% (95% CI 11%–37%) vs. lap 20% (12%–29%) | Indirect comparison of separately pooled proportions; overlaps primary cohorts listed here |
| Gianchandani [28] | Retrospective; single centre; Spain | 37 LT | Lap IPOM with tack fixation | 2/37 (5%); median FU 43 months (range 18–93); ascertainment: NR | Seroma 3, wound infection 1 | Single arm |

(*Continued*)

TABLE 1 Continued

| Study | Design and setting | Population | Repair (access; technique; mesh plane and material; closure) | Recurrence (n/N; follow-up; ascertainment) | Other principal findings | Main limitation |
|---|---|---|---|---|---|---|
| Cheema [29] | AHSQC registry; USA | 166 abdominal transplant recipients | Mixed | 21/75 (28%) composite at 2 years; registry follow-up; low follow-up rate | 30-day SSO/SSI 17/166 (10%) | Only 45% contributed to 2-year estimate |
| Woelfel [23] | ACHQC registry; USA | 165 SOT vs. 13,452 controls | Mixed; elective | 0.6% vs. 0.4% (p = 0.51); FU: NR; registry follow-up | Adjusted LOS 2.8 vs. 2.6 days (p = 0.5); SSI 2.4% vs. 4.0%; SSOPI 4.2% vs. 5.8% | Recurrence under-ascertained |
| Nielsen [30] | Single centre; prospectively maintained regional audit database; UK | 26 LT | Open peritoneal flap hernioplasty; retromuscular and intermuscular mesh (median 900 $cm^2$) | 0/26; median FU 54 months; clinical review and regional attendance records, imaging when indicated (postoperative CT in 3/26) | No infection | Specialist unit; single arm; selective imaging |
| Okumura [31] | Prospective cohort; single centre; USA | 38 SOT (79% LT); 33 elective | Open, mostly retrorectus after posterior component separation; biologic mesh | 3/38 (7.9%); FU: NR (protocol 3 years); ascertainment: NR | SSI 13.2%; unplanned reoperation 10.5% | Mixed organs; 5 non-elective; biologic mesh only |
| Wasielewski [3] | Retrospective; single centre; France | 141 LT with first open repair (of 803 LT) | Open; mesh 101 (intraperitoneal 65, retromuscular 36), suture 36 | 50/141 (35.4%); median 8.8 months to recurrence; cohort median FU 76 months from LT; clinical, CT if uncertain | Suture repair sHR 2.48 (1.25–4.90); sirolimus and MMF not associated | CT not systematic; retrospective |
| Hlavinka [32] | Retrospective review of prospective database; case-matched; single centre; USA | 47 SOT (31 LT, 11 kidney, 3 heart, 2 islet); 32 matched pairs | Mixed; absorbable 13, non-absorbable 34; bridged repairs included | Matched 4/33 vs. 1/33 (p = 0.16)†; mean FU 13 months; ascertainment: NR | Sirolimus 2/5, other 2/13, tacrolimus 0/29 (three-group p = 0.01); non-midline 3/12 vs. midline 0/31 (p = 0.02) | Denominator discrepancy†; small subgroups; mixed organs |
| Altolaguirre [14] | Retrospective; propensity-matched; single centre; USA | 42 LT vs. 105 non-transplant | Lap | No significant difference after matching (counts NR in abstract); median clinical FU 0.6 months; recurrence sought by electronic record review | Matched SSO 19.0% vs. 2.4% (p = 0.029) | Defect size not matched; very short clinical FU |
| Fowler [33] | Retrospective; single surgeon; USA | 36 LT | Open; biologic mesh (16; retrorectus 50%, intraperitoneal 37.5%) vs. no mesh [20]; mostly anterior component separation | 2/16 vs. 2/20 (p = 0.813); FU: NR (longer with mesh); ascertainment: NR | Infection 4/16 vs. 1/20 (p = 0.085); one death | Larger defects with mesh; small |

(*Continued*)

TABLE 1 Continued

| Study | Design and setting | Population | Repair (access; technique; mesh plane and material; closure) | Recurrence (n/N; follow-up; ascertainment) | Other principal findings | Main limitation |
|---|---|---|---|---|---|---|
| Banton [13] | Retrospective review of prospectively maintained series; single centre; USA | 51 LT (3 liver–kidney) | Robotic TAR; sublay synthetic mesh (median 832 $cm^2$) | 0/51; median FU 13.4 months (IQR 7.8–24.1); clinical examination, imaging in 10/51 | SSO 7.8%; no reoperation | Single arm; short FU; no routine imaging |
| Voidonikolas [12] | Single centre; prospective data for TAR, historical controls; USA | 53 LT | Open TAR via subcostal incision, retromuscular medium-weight polypropylene [23] vs. other techniques (30; 63% intraperitoneal) | 0/23 vs. 11/30 ($p < 0.01$); median FU 53 vs. 88.5 months; clinical ± CT/MRI | Unplanned reoperation 0% vs. 13.3% ($p = 0.03$) | Historical control; 4 vs. 14 surgeons; unequal FU |

*Not recommended; see Section *Access and intraperitoneal mesh*.

†Percentages in the source correspond to 33 patients per group, whereas the text describes 32 matched pairs. ACHQC, Abdominal Core Health Quality Collaborative; AHSQC, Americas Hernia Society Quality Collaborative; BMI, body mass index; CT, computed tomography; HPB, hepatopancreaticobiliary; IPOM, intraperitoneal onlay mesh; lap, laparoscopic; LOS, length of stay; LT, liver transplant; MMF, mycophenolate mofetil; MRI, magnetic resonance imaging; NR, not reported; sHR, subdistribution hazard ratio; SOT, solid organ transplant; SSI, surgical site infection; SSO, surgical site occurrence; SSOPI, SSO requiring procedural intervention; TAR, transversus abdominis release.

direction of selection bias is uncertain. With incomplete registry capture and underpowered single-centre comparisons, the data show numerically higher recurrence in some transplant cohorts but do not establish an independent transplant effect; no adequately adjusted estimate exists.

## Immunosuppression and mTOR inhibitors

Three questions should be kept separate: *de novo* sirolimus and healing of the transplant incision, maintenance immunosuppression and outcome after later repair, and changing immunosuppression around hernia surgery.

### De novo sirolimus and incision healing

In the only randomised trial, wound complications, including IH, occurred in 47% (30/64) of kidney recipients receiving sirolimus and 8% (5/59) receiving tacrolimus ($p < 0.0001$) [35], and sirolimus was associated with SSI in a cohort of 1,398 kidney recipients (odds ratio 3.21; 95% CI 1.26–8.21) [36]. The trial used trough targets of 10–20 ng/mL during early healing and does not estimate the effect of lower-dose maintenance therapy years later.

### Maintenance immunosuppression and repair outcome

This evidence is observational and small. Sirolimus was associated with recurrence after laparoscopic repair in 54 liver recipients ($p = 0.014$) [26], and mTOR inhibitor use with SSI in a Belgian series [25]. In the mixed SOT cohort, recurrence occurred in 2/5 patients taking sirolimus, 2/13 taking other regimens and 0/29 taking tacrolimus ($p = 0.01$ for the three-group comparison); both sirolimus recurrences followed complete fascial closure [32]. In contrast, neither sirolimus nor mycophenolate was associated with recurrence in 141 liver recipients [3]. Sirolimus is often prescribed after calcineurin inhibitor intolerance, renal impairment or malignancy [32], so confounding by indication is plausible, but its direction and magnitude are unknown. These findings should not be generalised to everolimus or to mTOR inhibition as a class. Indirect data from 732 patients immunosuppressed for any indication suggest that more than two agents and duration over 48 months predict complications [37].

### Perioperative conversion

Several centres convert sirolimus to a calcineurin inhibitor before repair [32, 38]. No study has measured its effect on wound complications or recurrence, or its risks of rejection, nephrotoxicity and loss of the benefit for which sirolimus was chosen.

## Operative approach and prosthesis

Repair strategies differ in five dimensions that are often conflated: access, release or reconstruction technique, mesh plane, prosthesis material, and fascial closure versus bridging (Table 1). Named techniques usually change several at once.

### Access and intraperitoneal mesh

Early comparative series favoured laparoscopic intraperitoneal onlay mesh (IPOM) repair, with no recurrence after 12 laparoscopic

repairs and recurrence or serious mesh infection after 7 of 15 open repairs in immunosuppressed, mostly liver recipients [21], and fewer minor complications (16.7% vs. 52.9%) and shorter stay than historical open controls in 35 liver recipients [27]. Single-arm laparoscopic series report recurrence of 5% at a median of 43 months [28] and 17% [26] in liver recipients, and similar recurrence in kidney or pancreas recipients and non-transplant controls [39]. A proportional meta-analysis of 546 mesh repairs after liver transplantation pooled complications of 23% (95% CI 11%–37%) for open and 20% (12%–29%) for laparoscopic repair [10]; overlapping intervals around separately pooled proportions do not establish equivalence. Laparoscopic and open repair have therefore been compared, but only against historical or unmatched controls. Robotic repair is reported only in small series [13, 40].

Subxiphoid defects lack cephalad fascia for fixation. Early laparoscopic series extended the mesh over the diaphragm with tack fixation beyond the defect [21]. This should not be done. Of 25 reported severe penetrating cardiac injuries after mesh fixation, 23 followed diaphragmatic tacks, 6 during repair of hernias extending to the subxiphoid region, and 12 patients died; manufacturers contraindicate tack deployment in the diaphragm, and tacks should not be used for diaphragmatic fixation above the costal arch [41]. Extraperitoneal dissection into the retroxiphoid space without costal or diaphragmatic fixation avoids this hazard [12], but has not been compared formally in transplant recipients.

### Extraperitoneal reconstruction

Extraperitoneal reconstructions report widely varying results. Peritoneal flap hernioplasty with large retromuscular and intermuscular mesh produced no recurrence or infection in 26 liver recipients at a median of 54 months [30]. Open TAR with retromuscular synthetic mesh through a midline incision in 44 liver recipients (median defect width 20 cm) was followed by recurrence in 11 (25%) at a median of 13 months, 8 from central mesh fracture [11]. In contrast, open TAR through the existing subcostal incision with medium-weight polypropylene mesh was followed by no recurrence in 23 liver recipients at a median of 53 months, compared with 11/30 (36.7%) after other, mostly intraperitoneal, techniques ($p < 0.01$) [12]. Comparators were historical, treated by 14 rather than 4 surgeons and followed for longer (median 88.5 months), so era, surgeon experience and technique cannot be separated. Robotic TAR in 51 liver recipients was followed by no recurrence at a median of 13.4 months [13], and biologic retrorectus reconstruction in 38 SOT recipients (79% liver) by recurrence in 7.9% [31]. These specialist series show what is achievable in selected patients; the contrast between the open TAR series suggests that incision, defect size and mesh may matter as much as the release.

### Mesh material, fascial closure and location

Associations with material are confounded by defect complexity, contamination, mesh plane and bridging. Recurrence was 3/13 with absorbable and 1/34 with non-absorbable synthetic mesh ($p = 0.06$) in the mixed SOT cohort [32]; not using synthetic mesh [25] and suture repair [3] were risk factors in liver series; and biologic mesh was associated with more infection than no mesh (4/16 vs. 1/20; $p = 0.085$), with similar recurrence, although it was used for larger defects [33]. None of these data establishes the superiority of a material. Location associations conflict: non-midline hernias recurred more often in the mixed SOT cohort (3/12 vs. 0/31; $p = 0.02$) [32], whereas midline location was a risk factor among immunosuppressed patients elsewhere [37], and neither study could adjust for case mix and technique together.

## Perioperative optimisation

Most modifiable risk factors are shared with the general hernia population. In univariable analysis, BMI >35 kg/m$^2$, diabetes and previous open abdomen were associated with SSO or SSI [29], and visceral obesity showed a trend towards recurrence (subdistribution HR 2.21; 95% CI 0.98–4.95) [3]. Weight reduction, smoking cessation, glycaemic control, nutritional assessment and multidisciplinary review of immunosuppression are reasonable before elective repair [7]. A London centre introduced a prehabilitation-based pathway after complications in 13 of 34 kidney recipients, including one death, and reported better hernia-related quality of life afterwards [42], but with a before-and-after design and 17 respondents its effect cannot be isolated.

Patients in the cohort linking lower intraoperative fluid with AKI [34] were not enrolled in protocolised volume restriction, so the association does not show that restrictive enhanced recovery protocols cause AKI or that liberal fluid prevents it. It supports baseline renal assessment, attention to calcineurin inhibitor levels, avoidance of dehydration and joint planning with the transplant team. AKI is systemic morbidity, not a measure of liver allograft dysfunction.

## Discussion

Three findings emerge, with different strengths of evidence. First, after selected elective repair, 30-day wound morbidity and length of stay are similar to those of non-transplant cohorts in most comparisons, although one matched series found more SSOs and AKI was common in one liver cohort. Second, recurrence ranges from 0% in selected specialist series to 35%–37% in larger cohorts with longer follow-up, but comparisons with non-transplant patients are few, unadjusted and underpowered. Third, *de novo* sirolimus impairs early incision healing in randomised data, whereas its association with recurrence after later repair rests on small, conflicting series. We identified no completed randomised trial of any repair or immunosuppression strategy in recipients with an established hernia.

Recent data refine earlier interpretations. The French cohort [3] and the AHSQC TAR series [11] temper the low recurrence of older laparoscopic and specialist series [21, 28, 30]. The contrasting TAR series [11, 12] show that a named technique is not a uniform intervention, so comparisons must specify incision, plane, mesh and closure. Registration of prophylactic mesh trials [15, 16] shows that prevention can be tested at transplantation.

TABLE 2 Principal areas of uncertainty in incisional hernia repair after abdominal organ transplantation.

| Question | Current evidence | Main limitation | Research priority |
|---|---|---|---|
| Does transplant status independently increase risk after repair? | Similar 30-day wound outcomes and LOS in most adjusted or matched comparisons; more SSOs after lap repair in one matched LT series; recurrence 0%–37% across transplant series, varying with selection, follow-up and ascertainment | Selection of fit patients (direction uncertain); incomplete registry recurrence capture; small, unadjusted recurrence comparisons | Registry analyses with transplant-specific fields, standardised recurrence ascertainment and adequate post-repair follow-up |
| Does immunosuppressive regimen affect outcome? | (a) RCT: *de novo* sirolimus increases transplant-incision wound complications; (b) small, conflicting series on maintenance sirolimus and recurrence after repair; (c) no data on perioperative conversion | Different exposures and settings; confounding by indication of unknown direction; very small sirolimus groups | Prospective data on regimen, reason for regimen and changes around surgery; establish equipoise and feasibility before a conversion trial with wound, recurrence and graft-safety outcomes |
| Which access and reconstruction? | Lap IPOM vs. open compared only with historical or unmatched controls; extraperitoneal reconstructions report recurrence of 0%–25% in single-arm or historically controlled series | Access, release, mesh plane, material and closure vary together; era, incision and surgeon experience confounded | Prospective comparisons of fully specified strategies, including robotic extraperitoneal repair |
| Which mesh and closure? | Higher recurrence with suture repair and, in small series, absorbable or non-synthetic mesh; more infection with biologic mesh in one series | Small numbers; mesh choice confounded by defect complexity, contamination, plane and bridging | Material-, plane- and closure-specific outcome reporting |
| How should subxiphoid defects be fixed? | Penetrating cardiac injury and death after diaphragmatic tack fixation (case series, mostly non-transplant) | Incidence unknown; no comparison of alternative fixation in transplant recipients | Avoid diaphragmatic tacks; report fixation method |
| Does hernia location matter? | Conflicting associations for midline and non-midline defects | Different case mix and techniques; no adjustment for both | Location-stratified analyses using EHS classification |
| Can hernia be prevented at transplant closure? | SSI, obesity and incision type are consistent risk factors; two RCTs of prophylactic mesh registered (liver: Terminated after 36 patients, no results; kidney: Recruiting) | No published trial results; non-transplant evidence may not transfer | Completion and reporting of registered trials; trials in high-risk recipients |
| What are the systemic renal risks? | AKI in 48% of one liver cohort (creatinine rise >0.3 mg/dL within 24 h); associated with lower intraoperative fluid | Single cohort; definition-specific; observational fluid association | Prospective renal outcome reporting with standard AKI definitions |
| What are the graft-specific risks? | Allograft function and rejection after repair rarely reported | Outcomes not collected | Core outcome set including graft and patient-reported outcomes |

AKI, acute kidney injury; EHS, European Hernia Society; IPOM, intraperitoneal onlay mesh; lap, laparoscopic; LOS, length of stay; LT, liver transplant; RCT, randomised controlled trial; SSI, surgical site infection; SSO, surgical site occurrence.

## Controversies

Whether transplantation confers risk beyond comorbidity remains unresolved: registry data suggest that comorbidity drives outcome [23, 24, 29], whereas single-centre data suggest excess recurrence [32], and both are compatible with incomplete registry follow-up and underpowered cohorts. Converting sirolimus before repair is biologically plausible but carries risks of rejection and nephrotoxicity and is unproven; it requires joint decision-making, and patients unsuitable for conversion should not be denied repair. Comparisons of operative strategies are confounded by era, defect size, incision, surgeon experience and case selection, whose directions are not established, although in the largest recent comparison the newer technique was performed by fewer, specialised surgeons [12]. Whether intraperitoneal mesh complicates re-transplantation is unquantified.

## Research gaps

The main gaps are summarised in Table 2. Recurrence is defined inconsistently and post-repair follow-up is often 2 years or less, although median time to recurrence after repair was 8.8 months in one cohort [3] and 29 months in another [12]. Graft function and rejection are seldom reported, and patient-reported outcomes after repair come from a single pilot study (16 of 24 liver recipients

responding) [43]. Pancreas and combined-organ recipients appear mainly within mixed cohorts [1, 39]. UK data are limited to two single-centre series [30, 42], so applicability to NHS practice, including variation in immunosuppression protocols and access to specialist abdominal wall services, is uncertain.

Prevention at transplant closure is distinct from treatment of an established hernia. The PRINC trial of onlay poly-4-hydroxybutyrate mesh after liver transplantation [15] is listed on ClinicalTrials.gov (NCT03222102) as terminated after enrolling 36 patients, with no results posted. A kidney transplant trial of onlay self-gripping polyester mesh, with computed tomography at 24 months as primary outcome [16], is listed as recruiting, with primary completion expected in December 2026 (NCT04794582). Evidence on prophylactic mesh in transplant recipients is therefore absent rather than negative, and trials after midline laparotomy in non-transplant patients may not transfer to immunosuppressed recipients with transverse or iliac fossa incisions.

## Limitations of this review

This is a narrative mini review. Study selection was not duplicated and no protocol was registered, and no formal risk-of-bias assessment or meta-analysis was performed, so selection and emphasis reflect author judgement. Restriction to English-language published studies may have omitted relevant data. Most included studies are retrospective single-centre cohorts with heterogeneous definitions of SSO, SSI and recurrence, so we report study-specific estimates rather than pooled rates. Several sources combine organ types or include non-transplant immunosuppressed patients, and some figures come from internally inconsistent primary reports (Table 1).

## Future directions

Transplant-specific fields (organ, immunosuppressive agents and levels, changes around surgery, graft function, rejection and AKI) could be added to existing hernia registries, with standardised recurrence ascertainment, patient-reported outcomes and long post-repair follow-up. Before any trial of perioperative sirolimus conversion, prospective data should establish whether equipoise exists, because sirolimus is often chosen for renal protection or malignancy. A trial would need to restrict eligibility to recipients in whom calcineurin inhibitor exposure is acceptable, record the reason for the existing regimen, measure early wound complications, recurrence and graft safety, and recruit across centres. Comparisons of fully specified extraperitoneal reconstructions with laparoscopic or robotic alternatives, and reporting of the registered prophylactic mesh trials, are further priorities.

In summary, selected, optimised recipients undergoing elective repair appear to have short-term wound morbidity similar to that of non-transplant patients in most comparisons, although AKI is frequent. Recurrence is common, but whether transplantation independently increases it is unknown. Evidence for sirolimus conversion, particular reconstructions and mesh materials is of low certainty, and pancreas-specific data are sparse. Diaphragmatic tack fixation should be avoided. The evidence does not yet support firm practice recommendations.

# Author contributions

KV: Conceptualisation, Methodology, Investigation, Data curation, Formal analysis, Visualisation, Writing – original draft, Writing – review and editing. MP: Data curation. FK: Conceptualisation, Methodology, Validation, Supervision, Writing – review and editing. VP: Conceptualisation, Validation, Supervision, Writing – review and editing. All authors contributed to the article and approved the submitted version.

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