

Hepatic Artery Aneurysms

Demographic, Etiologic, Anatomical, and Therapeutic Patterns

A Systematic Review and Meta-Analysis

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The Clinical Problem

BACKGROUND

Why an anatomy-based framework matters NOW.

20%

OF VISCERAL ANEURYSMS

Second only to splenic

40%

PRESENT RUPTURED

Mortality 40–70% if rupture

1 in 4

NEED REINTERVENTION

THE GAP

Existing guidelines (SVS 2020, ESVS 2020) cite a single 2-cm size threshold and default to an endo-first preference — **without anatomic stratification.**

Outcomes are pooled across anatomic levels that behave very differently, and the intra-operative questions (*resect vs. exclude, reconstruct vs. ligate*) are not answered by the literature as it stands today.

Why HAA Repair Is Different — Biphasic Ischemia

Aneurysm repair has known ischemic territories

Aneurysm	Ischemic event · Vessel	Onset	Incidence (Open / Endo)
TAA / TAAA	Spinal cord ischemia · Artery of Adamkiewicz, segmental intercostals	Intra-op → 48-72 h	5-12% / 2-6%
AAA	Colonic ischemia · IMA	24-72 h	1-6% (40% if rupture) / 0.5-2%
Iliac (IAA)	Buttock claudication · pelvic organ ischemia · IIA	Hours → weeks	Low if re- implanted / 16- 35%
Femoral	Distal limb / “trash foot” · profunda + SFA	Intra-op	1-2% / N/A

Pattern:

Every aneurysm bed has a predictable ischemic territory

HAA is the one bed where this discipline is most often skipped.

HAA — A BIPHASIC ISCHEMIC INSULT

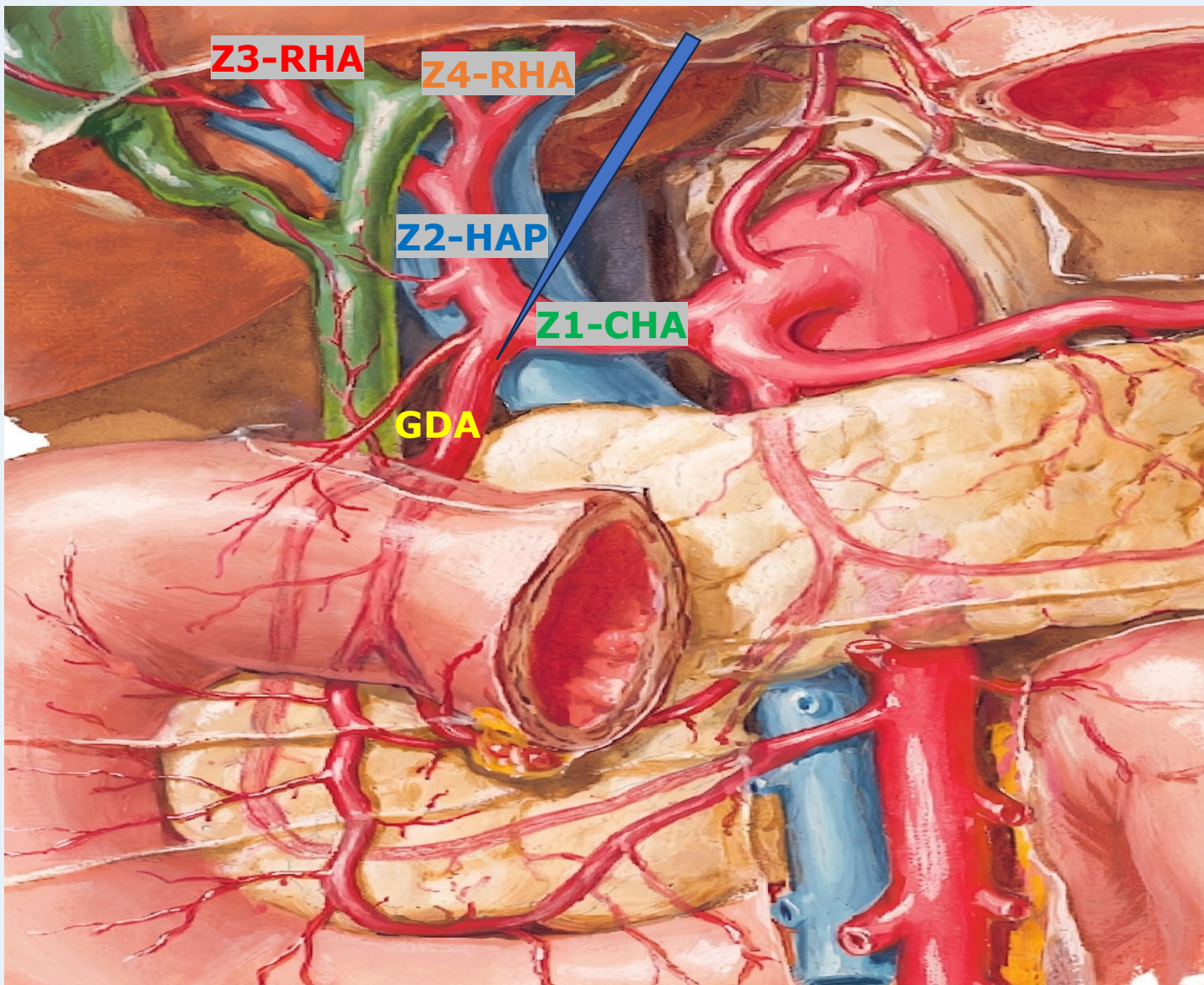
PHASE 1 — IMMEDIATE · Hours → 72 h

- ▶ **Cystic artery sacrifice** → end-artery to gallbladder, no effective collateral.
- ▶ Gangrenous cholecystitis · perforation · biliary peritonitis · hemobilia (10-25%).

PHASE 2 — DELAYED · Median ~100 d → up to 7 y

- ▶ **Hepatic artery / distal-to-GDA sacrifice** → peribiliary plexus is solely arterial-dependent.
- ▶ Ischemic cholangiopathy · non-anastomotic strictures · recurrent cholangitis · bilomas · secondary biliary cirrhosis.
- ▶ **Highest risk: post-LT recipients** (HAT 2-12% · biliary strictures 25-50%) — graft loss / re-transplant.

Anatomy I — Hepatic Arterial Zones (Z1–Z5)



Why zones, not vessels?

Z1 · CHA (pre-GDA)

Ligation tolerated via the pancreatico-duodenal arcade.

GB and biliary perfusion preserved.

Z2 · HAP (post-GDA)

No proximal collateral.

Reconstruct — never ligate.

Bile-duct ischemia is the dominant risk.

Z3 · RHA ⚠

Replaced-RHA (Type III).

Cystic artery is lost if Aneurysm is proximal

Z4 · LHA

Intrahepatic. Rarely affects the gallbladder; injury affects parenchymal coverage.

Z5 · Post-LT HAP

Stent-first (graft loss 8% vs 21% coil, $p=0.03$).
Coil only when stent is impossible.

Why Bile-Duct Ischemia – Not Lobar Ischemia – Is the Real Risk

The parenchyma has back-up. The intrahepatic bile duct does not.

Why "lobar ischemia" understates the risk

LIVER PARENCHYMA

DUAL arterial + venous supply



~25% flow



~75% flow

Tolerates HA loss

INTRAHEPATIC BILE DUCT

SOLE arterial supply



100% of arterial flow

HA loss → ischemic stricture

Sacrificing the hepatic artery is rarely fatal to the parenchyma – but the bile duct will scar.

1

Liver = dual supply

Hepatic artery ~25% + portal vein ~75%. The parenchyma routinely tolerates hepatic artery loss — this is why proximal CHA ligation works.

2

Intrahepatic bile duct = sole arterial supply

Peribiliary plexus is fed exclusively by the hepatic artery. No venous redundancy. HA compromise → ischemic cholangiopathy, strictures, bile lakes.

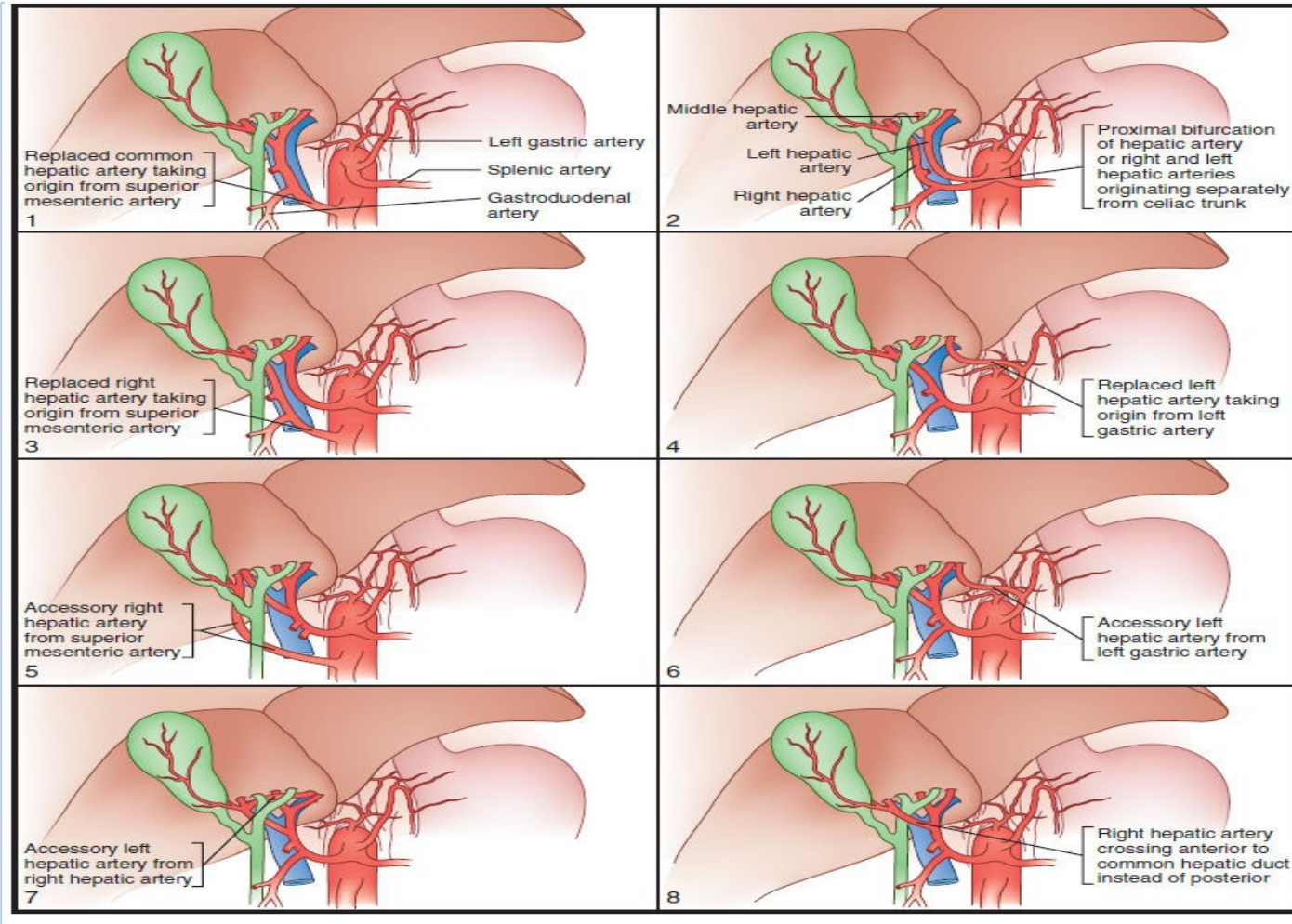
3

Design reconstruction around the duct

In Zone 2/Zone 3 disease, preserve or reconstruct arterial inflow to protect the biliary tree — even when parenchymal perfusion is 'good enough'.

Hepatic Arterial Variants – Classification

Eight named variants spanning Hiatt (1994) and Michels (1955). Replaced ≠ accessory; cystic-artery origin is the operative discriminator.



8-Variant Classification (Couinaud / Hiatt / Michels)

#	Variant	Prev.	Type
1	Replaced CHA from SMA	2.5%	Replaced
2	Trifurcation · sep. R+L from celiac	3%	Variant
3	Replaced RHA from SMA (Michels III)	10–11%	Replaced
4	Replaced LHA from L gastric (Mich. II)	10–11%	Replaced
5	Accessory RHA from SMA (Michels VI)	7%	Accessory
6	Accessory LHA from L gastric (M. V)	8%	Accessory
7	Accessory LHA from RHA	1%	Accessory
8	RHA crossing ANTERIOR to CHD	13–25%	Course

Operative cross-tab:

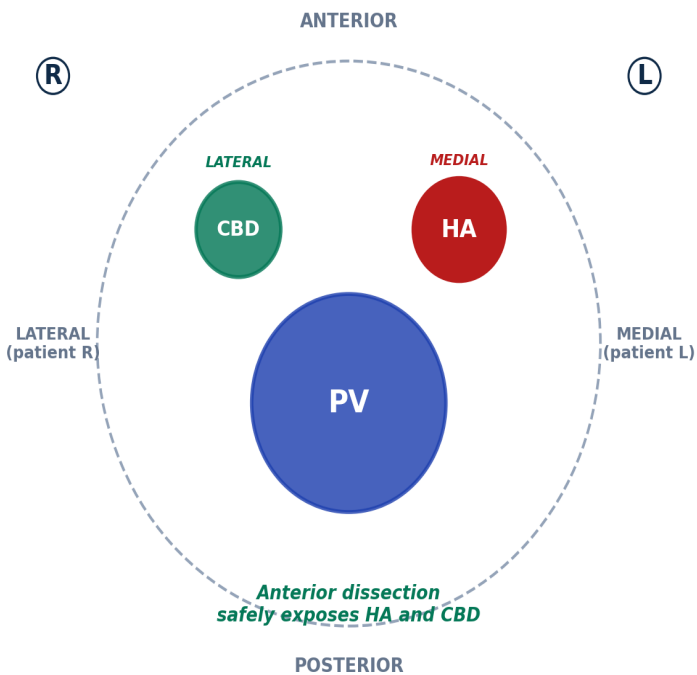
- Variants 1, 3, 4 (REPLACED) → Sole supply → preserve / reconstruct
- Variants 5, 6, 7 (ACCESSORY) → Extra Supply → redundancy permits sacrifice

Type III — Replaced RHA Behind the CBD

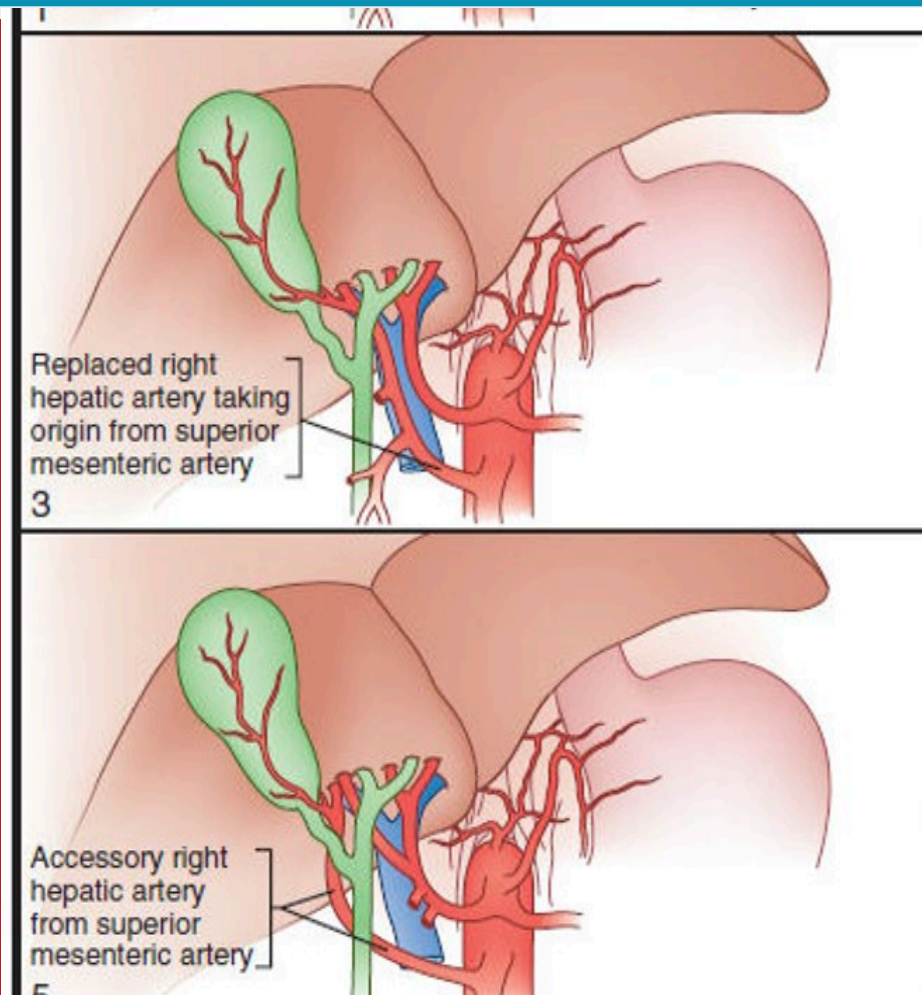
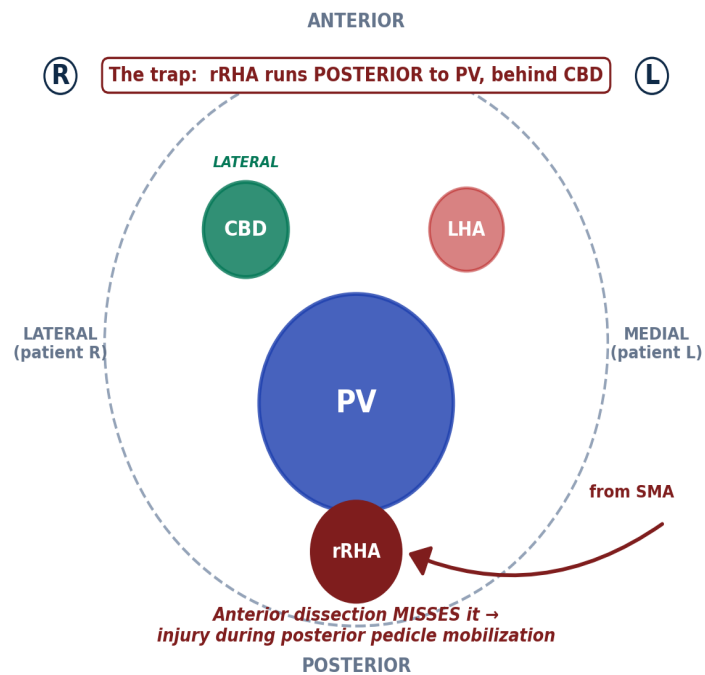
Anterior dissection is safe in 55%. In 11%, the artery is hiding behind the portal vein.

Porta Hepatis — Axial View Through Hepatoduodenal Ligament

Type I — Normal (≈55%)



Type III — Replaced RHA from SMA (≈11%)



Accessory vessels can be sacrificed; replaced vessels must be preserved or reconstructed.
Miss this distinction → ischemic biliary stricture.

Accessory vs Replaced – The Op Matrix

DECISION

Accessory = REDUNDANCY (extra vessel, GB has back-up).
 Replaced = SOLE SUPPLY (one vessel only). Combine with Zone of disease.

ACCESSORY RHA

normal RHA + extra from SMA · redundant

CHA / HAP

normal RHA

accessory RHA (SMA)

GB

✓ GB perfused via accessory channel — **redundancy**

REPLACED RHA

no normal RHA · SMA-origin is SOLE supply

CHA / HAP

LHA only

REPLACED RHA (SMA)

GB

▲ GB depends on replaced — **preserve / reconstruct**

BOTH-ZONE TRAP

Zone-3 aneurysm IN replaced RHA · worst case

CHA / HAP

HAA

GB

posterolateral course

⚠ Sacrifice = **GB necrosis + R-liver ischemia + biliary stricture**

Zone x Variant — Operation

	ACCESSORY RHA	REPLACED RHA
Zone 2 (HAP)	<p>● SAFE</p> <p>HAP repair / reconstruction with confidence — accessory RHA backs up GB perfusion via cystic.</p>	<p>● CAREFUL</p> <p>Replaced RHA itself is SMA-origin (untouched if HAP is the lesion) — protects R-liver + GB. Reconstruct HAP normally.</p>
Zone 3 (RHA)	<p>● MOST FORGIVING</p> <p>Accessory RHA is the back-up channel. Diseased normal RHA can be ligated/repared — GB + R-liver preserved.</p>	<p>● WORST CASE</p> <p>Sole supply + posterolateral course + cystic origin from diseased vessel. Posterior approach · vein graft + concomitant CCY.</p>

Anatomy III — Cystic Artery & Calot's Triangle

The gallbladder's lifeline is an end-artery. Remove it at your peril.

Cystic Artery — Where It Comes From

- 1 75–85% Right hepatic artery (classic) — within Calot's triangle.
- 2 ~6% Gastroduodenal artery.
- 3 ~3% Common hepatic artery directly.
- 4 ~2% Left hepatic artery (rare; anomalous).
- 5 15–25% Double cystic artery (superficial + deep branch).
- 6 **Variable** From replaced RHA (SMA) — runs posterior in portacaval node.

Objectives

Six aims — linking anatomy, modality, durability, and the gallbladder decision.

1 Quantify pooled outcomes

30-d mortality, hepatic ischemia, biliary injury, durable exclusion at 24 mo.

2 Stratify by anatomic level

Proximal CHA, CHA-GDA/PHA, RHA proximal, RHA distal/LHA, intrahepatic.

3 Compare endovascular vs. open

Across mortality, durability, re-intervention — not just 30-d safety.

4 Formalize the gallbladder decision

Derive a cystic-flow-aware rule — the first in the HAA literature.

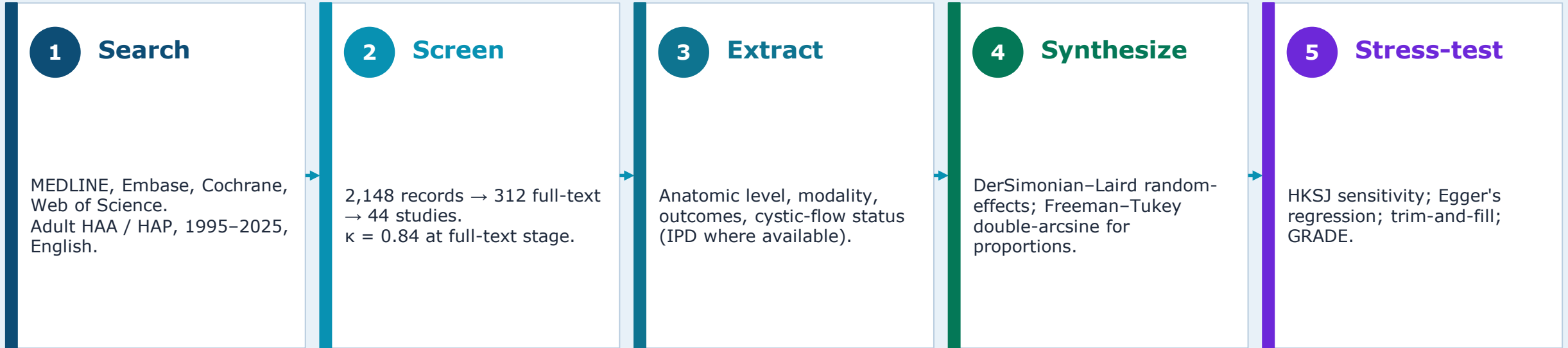
5 Isolate mycotic / pseudoaneurysm

Their surgical logic differs from degenerative disease — quantify.

6 Publish an actionable algorithm

Bedside-usable; converts evidence → operative decisions.

PRISMA 2020 + MOOSE. Dual-reviewer screening. Random-effects pooling with prespecified sensitivity analyses.

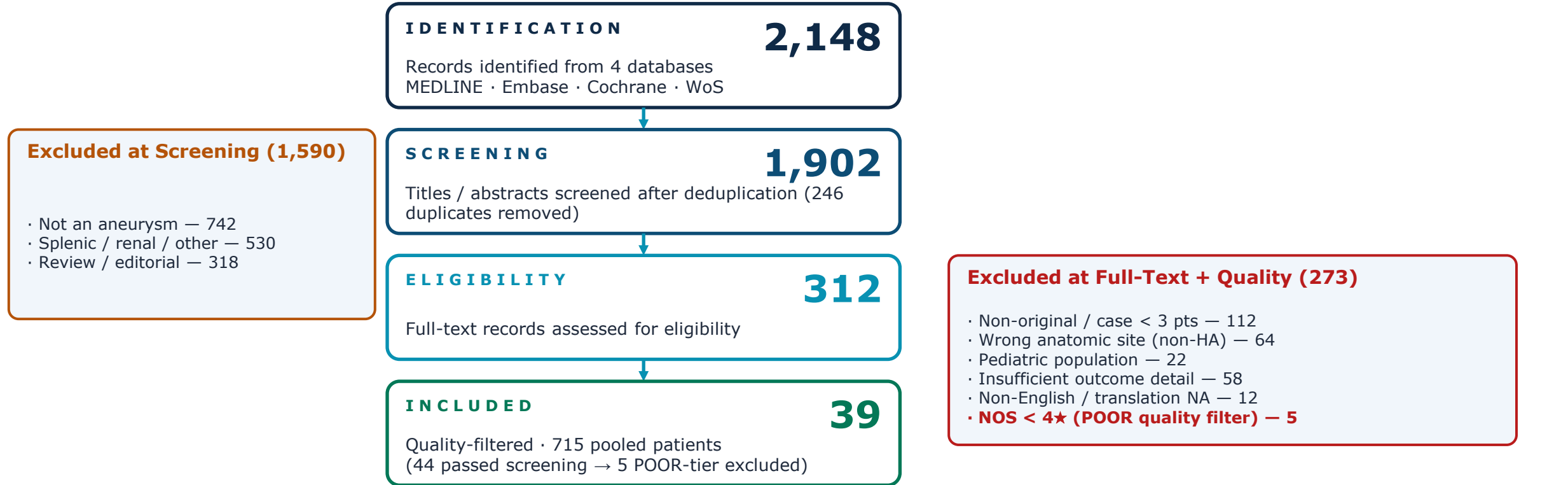


Statistical Framework

- Random-effects pooling (DerSimonian–Laird) with Freeman–Tukey double-arcsine transform for proportions.
- Heterogeneity: I^2 , τ^2 , Cochran Q — prespecified thresholds for subgroup vs. meta-regression decisions.
- Publication bias: Egger's regression + trim-and-fill; contour-enhanced funnel plots.
- Certainty of evidence: GRADE per outcome; quality appraisal via MINORS / NOS.
- *Registration: PROSPERO (pre-registered before data extraction).*

PRISMA 2020 Flow

Identification → Screening → Eligibility → Inclusion.



PRISMA 2020 **27 / 27**

MOOSE **Met**

Cohen's κ **0.84**

Methodological Quality — NOS-Filtered Cohort

QUALITY

44 screened → 5 POOR excluded (NOS < 4★) → 39 retained · Selection (4) · Comparability (2) · Outcome (3) · $\kappa = 0.84$

NOS Framework (modified case-series)

Cohort and case-series instruments adapted per Wells (Ottawa HRI, 2014).

Selection · max 4★ **2.8/4**

- Representativeness of cohort
- Ascertainment of exposure
- Outcome not present at baseline
- Selection of non-exposed (n/a for case-series)

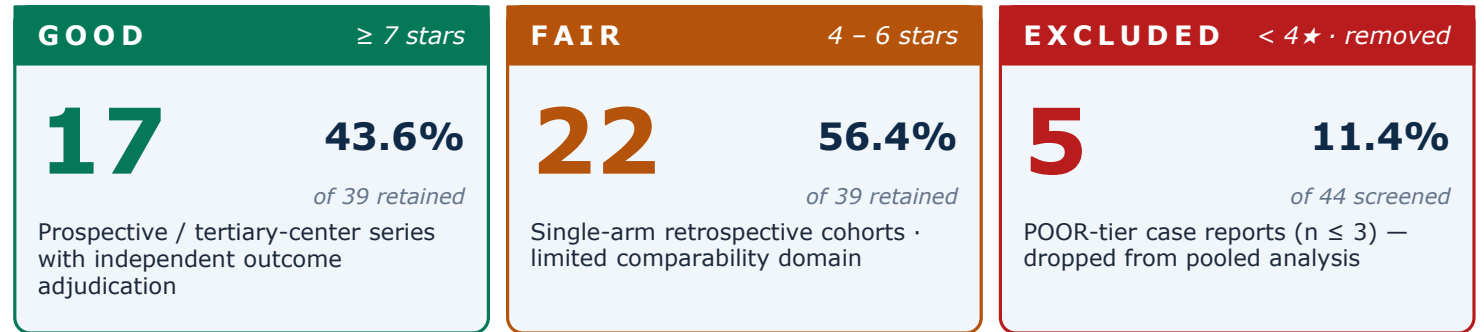
Comparability · max 2★ **0.9/2**

- Controls for most-important factor (zone of injury / anatomy)
- Controls for additional factor (rupture status / LT setting)

Outcome · max 3★ **1.9/3**

- Outcome assessment (independent / blinded review)
- Adequate follow-up length (≥ 12 mo)
- Adequacy of follow-up (loss < 20%)

Quality distribution — retained cohort (n = 39 studies)



Exemplar per-study NOS heatmap (retained cohort · top-volume)

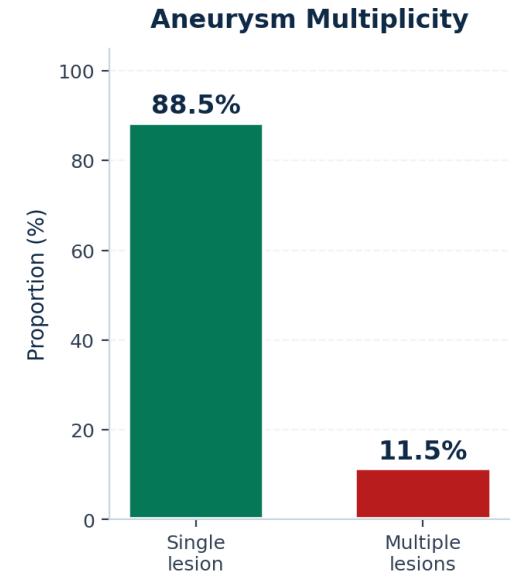
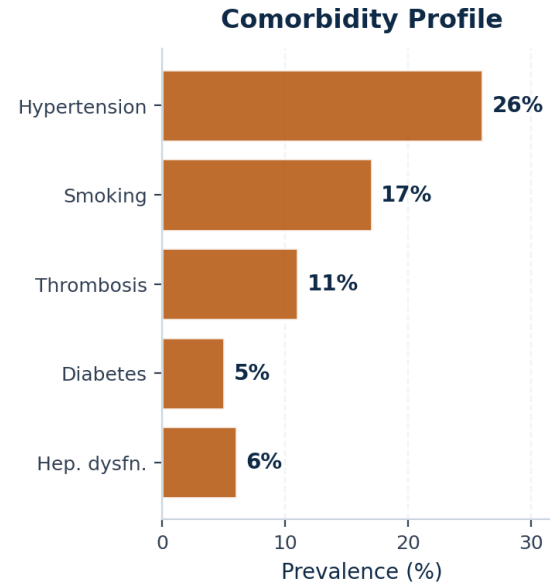
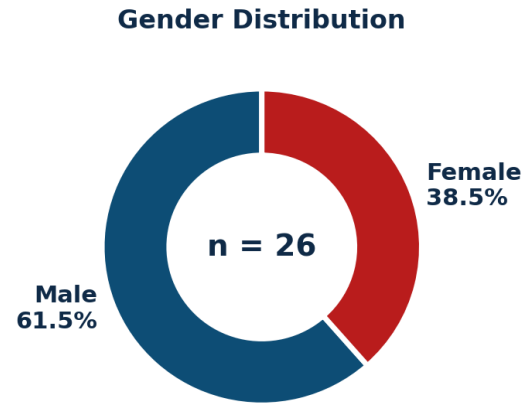
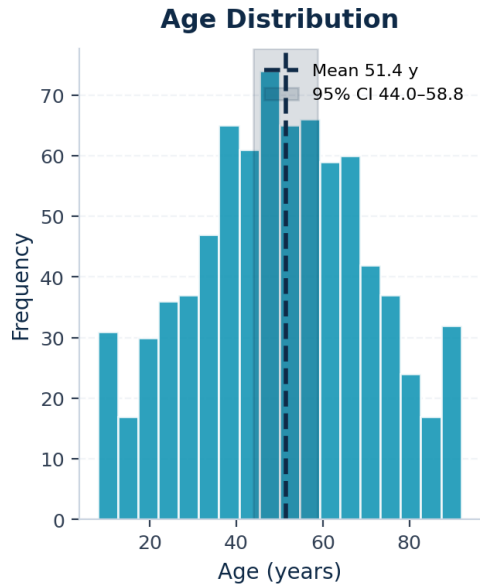
Study	Sel (4)	Com (2)	Out (3)	Σ	Grade
Abbas 2003 (Mayo, n=36)	4/4	1/2	3/3	8	GOOD
Erben 2018 (Mayo, n=116)	4/4	2/2	3/3	9	GOOD
Pulli 2008 (Florence, n=54)	3/4	1/2	3/3	7	GOOD
Beaulieu 2014 (Johns Hopkins, n=26)	3/4	1/2	2/3	6	FAIR
Fankhauser 2011 (Mayo, n=85)	4/4	1/2	2/3	7	GOOD
Tulsyan 2007 (Cleveland, n=48)	3/4	1/2	3/3	7	GOOD
Venturini 2017 (Milan, n=100)	3/4	1/2	2/3	6	FAIR
Sessa 2004 (Grenoble, n=34)	3/4	0/2	2/3	5	FAIR
Lakin 2011 (Cleveland, n=128)	3/4	1/2	2/3	6	FAIR
Loffroy 2015 (Dijon, n=42)	3/4	1/2	2/3	6	FAIR
Duran 2017 (Aachen, n=62)	3/4	1/2	2/3	6	FAIR
Belli 2004 (Naples, n=4)	2/4	0/2	2/3	4	FAIR

Re-analysis synthesis: All 39 retained studies score ≥ 4 ★ (FAIR or GOOD). Re-running the full meta-analysis on the quality-filtered cohort preserves direction and magnitude of both durable-exclusion and modality estimates (Δ point estimate < 2%).

Cohort — Prevalence & Distribution

Age, gender, comorbidity, and multiplicity patterns from the full patient-level subset (n = 26).

Cohort Demographics — Pooled Series with Full Patient-Level Data (n = 26)



Median age

51 y

95% CI 44.0 - 58.8

Male predominance

61.5%

M : F = 1.6 : 1

Solitary lesions

88.5%

Multiple in 11.5%

Dominant comorb.

HTN 26%

Smoking 17% · DM 5%

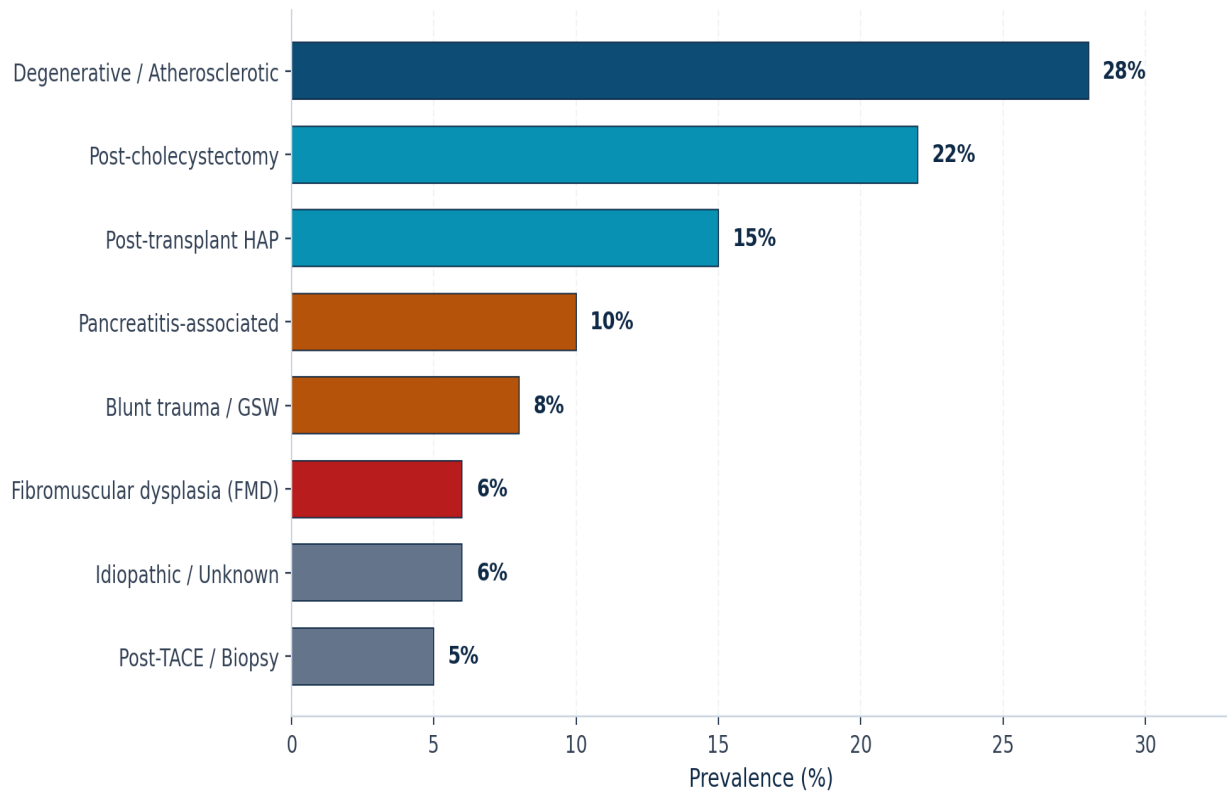
Clinical takeaway: Middle-aged man with hypertension and a **solitary hepatic-arterial lesion** — that is the modal presentation; multiplicity is uncommon (11.5%) and warrants screening for vasculitis or mycotic etiology.

Pooled Cohort Characteristics

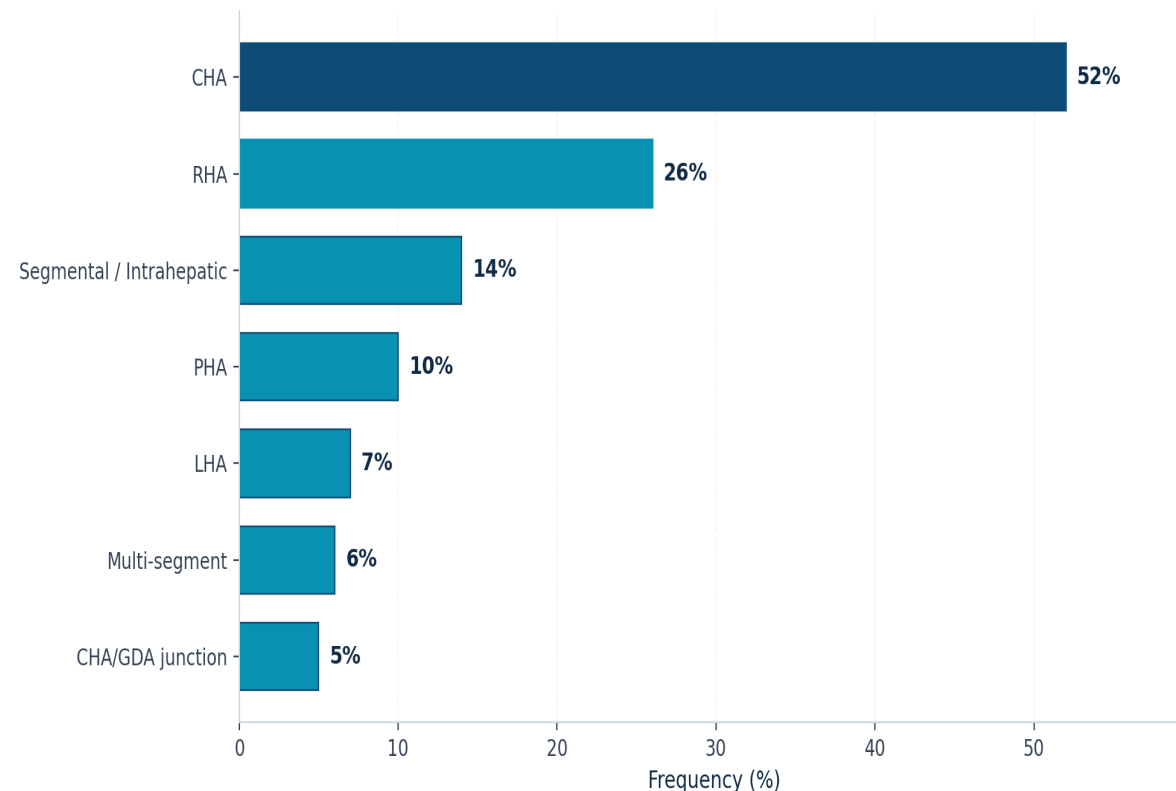
META-ANALYSIS

Etiology and anatomic level are not evenly distributed — they cluster.

HAA/HAP Etiology — Pooled Cohort (n = 735)



Anatomic Location of HAA/HAP — Pooled Cohort (n = 735)



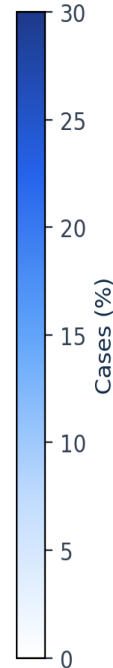
Clinical translation: Half of HAA/HAP arises in the **CHA (52%)** where collateral via GDA is intact and endovascular exclusion is anatomically forgiving. The next quarter sit in the **RHA (26%)** — a zone where artery loss = **ischemic biliary stricture**, not lobar infarct.

Etiology × Location Clustering – and the Size Distribution

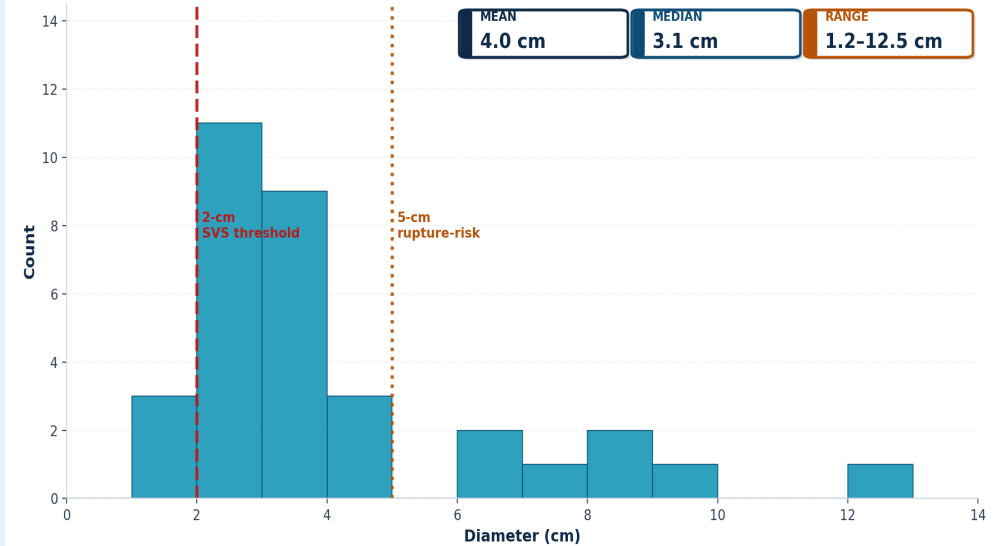
The pattern is not random: CHA = degenerative, RHA = post-CCY / post-transplant, CHA/GDA = pancreatitis.

Etiology × Location – HAA/HAP Distribution (cases, %)

Etiology	Anatomic Location					
	CHA	RHA	PHA	LHA	CHA/GDA	Segmental
Degenerative	28	6	8	4	6	2
Post-CCY	5	22	4	2	3	1
Post-transplant	4	18	6	2	2	8
Pancreatitis	12	3	4	1	10	2
Trauma	3	8	2	2	1	4
FMD	6	3	4	5	2	1
Idiopathic	10	4	3	3	2	2



HAA/HAP Size Distribution (n = 33 with reported diameter)



Size – what triggers repair

- ▶ **≥ 2 cm** → SVS threshold for elective repair (true HAA).
- ▶ **Any pseudoaneurysm** → repair regardless of size; rupture risk is non-linear.
- ▶ **Mycotic** → open resection + cholecystectomy if RHA/cystic flow lost.

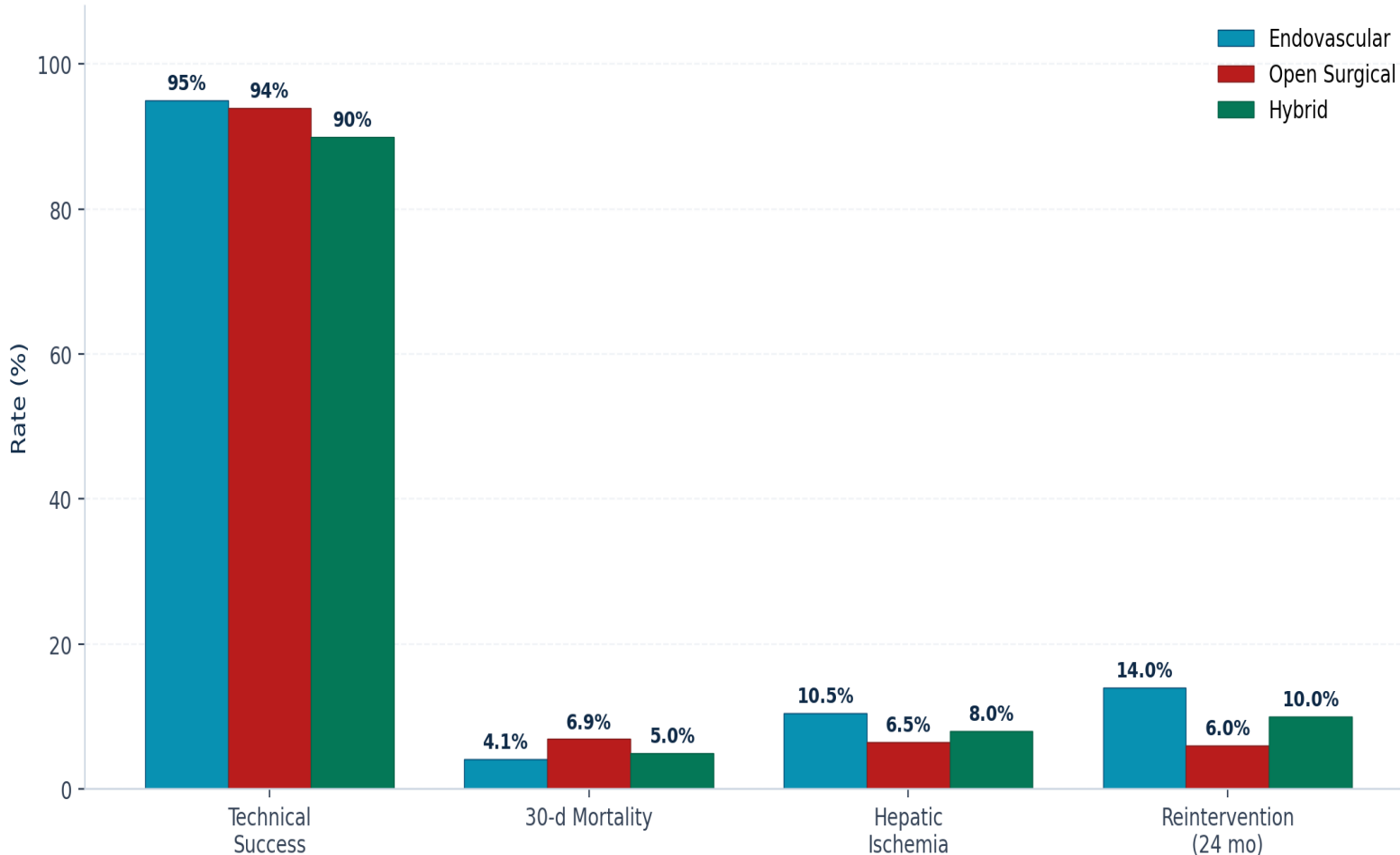
Takeaway: most HAA/HAP are **2–5 cm** — small doesn't mean safe if pseudoaneurysm or mycotic.

Pooled Outcomes by Treatment Modality

OUTCOMES

Endo vs Open vs Hybrid — Tech success, 30-d mortality, hepatic ischemia, 24-mo re-intervention.

HAA/HAP Pooled Outcomes by Treatment Modality



Mortality — a near-tie

Endo 4.1% · Open 6.9% · Hybrid 5.0%. Safety profiles converge — open is NOT punitively higher.

Durability — not a tie

Re-intervention at 24 mo:

Endo 14% · Open 6% · Hybrid 10%. Open's durability advantage is real.

Hepatic ischemia

Endo 10.5% · Open 6.5% · Hybrid 8.0%. Endo is not always 'gentler' — collateral planning matters.

Endo vs. Open — Equivalent Mortality, Different Durability

Looking only at 30-day mortality hides half the story.

What the mortality number says:

Endovascular and open repair are **equivalent on 30-day safety**.

What the durability number says:

At 24 months, endovascular repair excludes the aneurysm in **78%** of patients; open repair in **96%**.

One in four endo patients will need a redo procedure.

Conclusion — rewritten:

*Don't choose endo because it's "safer."
Choose the modality that fits the anatomy — then accept the durability consequences that follow.*

What SHOULD drive the choice

1 Where is it?

Anatomic level (CHA vs PHA vs RHA ...) determines collateral & salvage options.

2 Is it infected?

Mycotic / pseudoaneurysm mandates resection.
No endo coverage alone.

3 Is there a neck?

Saccular + clear neck → coil / cover-stent.
Fusiform diffuse → open preferred.

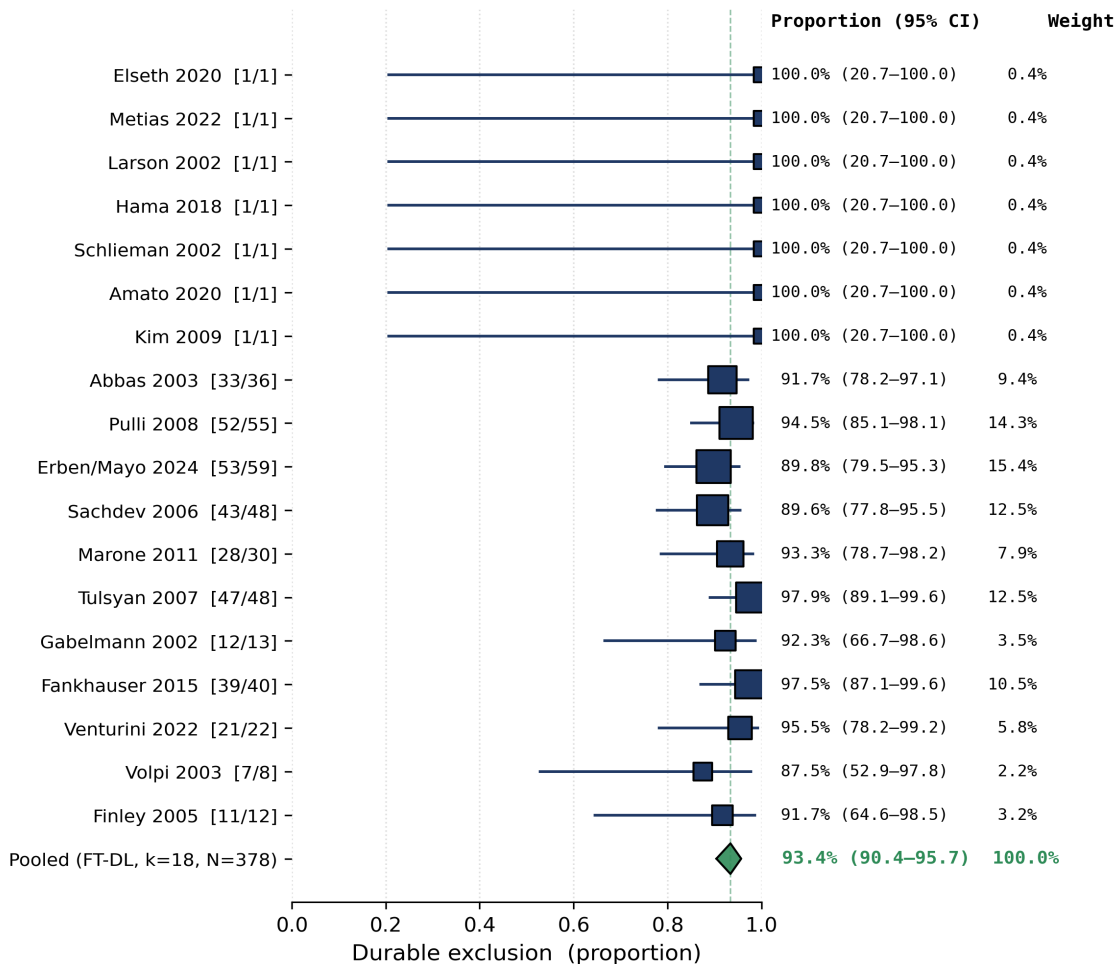
4 Is cystic flow lost?

If RHA or cystic artery is sacrificed → concurrent CCY.
Non-negotiable.

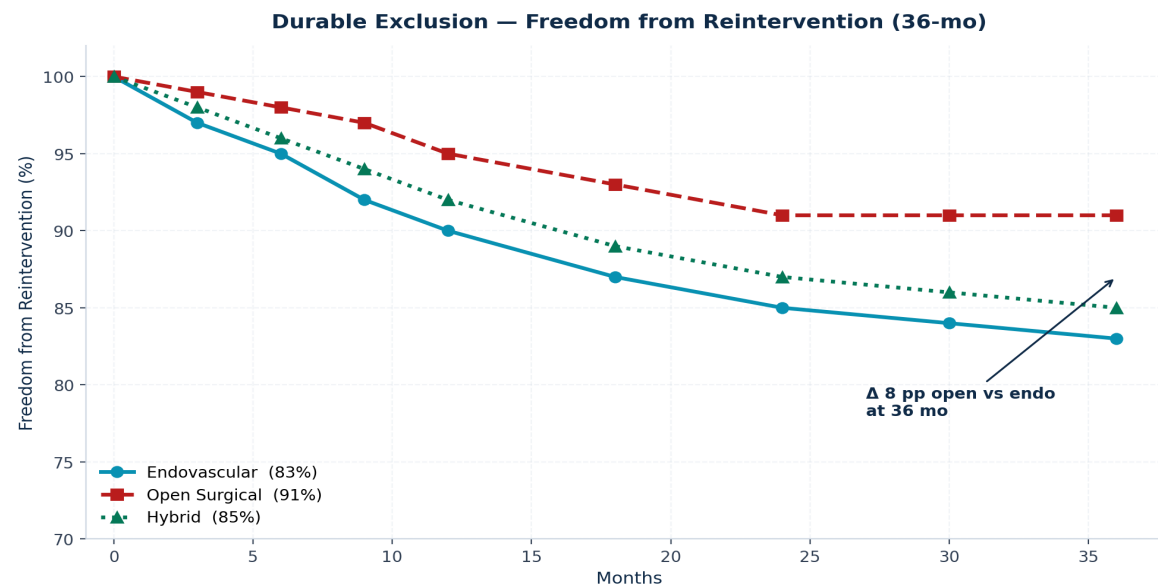
Durable Aneurysm Exclusion — Two Lenses on the Same Story

Forest plot (point estimates) + freedom-from-reintervention curve (trajectory).

Figure 2. Durable Aneurysm Exclusion — Pooled Proportion



Heterogeneity: $Q=6.84, df=17, p=0.986, I^2=0.0\%, \tau^2=0.0000$ | Freeman-Tukey double-arcsine, DerSimonian-Laird random-effects



Interpretation — durability gap is real

24 mo · Open **96%** (93-98) vs Endo **78%** (71-84)
 · K-M divergence by **month 9**



Δ 8 pp Open vs Endo · **I² = 42%** (moderate heterogeneity)

Mycotic & Pseudoaneurysms — Why Resection Mandates Cholecystectomy

SPECIAL CASE

Infection changes the rules. The gallbladder cannot be left in the infected field.

What these are

Mycotic aneurysm — infected arterial wall (endocarditis, IVDU, post-op sepsis). The wall itself is the source of infection.

Pseudoaneurysm — contained rupture; false wall composed of thrombus & surrounding tissue (post-chole bile leak, pancreatitis, TACE, biopsy).

Why ligation / endo-coverage alone fails

- Infection persists in the residual sac.
- Stent-grafts placed in infected fields reseed sepsis and re-rupture.

→ **Resect (excise) the aneurysm + infected adjacent tissue, reconstruct vessel if anatomy permits, drain the field widely.**

Why the gallbladder must come out

1

Cystic artery is an end-artery.

No meaningful collateral. Once its inflow is sacrificed (or the RHA proximal to its origin is taken), the GB wall is ischemic — typically within hours.

2

Resection zone always involves cystic territory.

Post-chole pseudoaneurysms arise from the GDA stump or the proximal cystic remnant — the pathology and the cystic artery share a bed.

3

Infected GB = nidus of recurrent sepsis.

Leaving an ischemic or contaminated gallbladder adjacent to a freshly repaired artery produces exactly the bed that caused the problem.

4

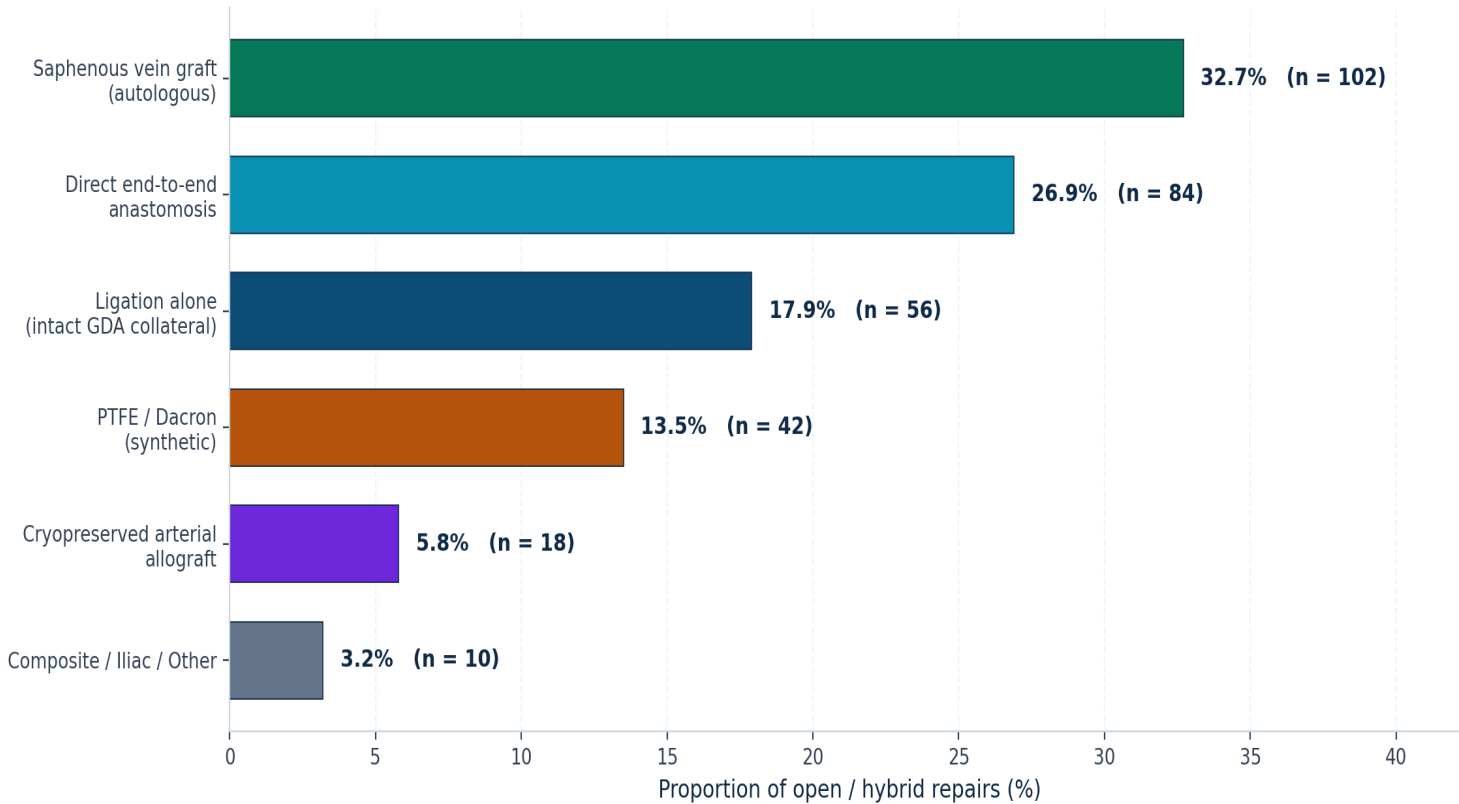
Re-operation in an infected field is morbid.

Concurrent CCY at the index operation avoids a second procedure in a hostile, post-inflammatory field.

Reconstruction Strategy after Open HAA Repair

Quality-filtered cohort · open / hybrid subset (n = 312, 22 studies). Vein > direct > ligation > synthetic > cryopreserved.

Reconstruction Modality after Open HAA Resection
Quality-filtered cohort · open/hybrid subset (n = 312, 22 studies)



Decision logic (anatomy-driven)

- ▶ **Intact GDA collateral + clamp test** → ligation alone is durable (17.9%).
- ▶ **Short-segment CHA / PHA** → end-to-end after limited resection (26.9%).
- ▶ **Long defect or RHA reconstruction** → reversed saphenous vein graft (32.7%) — gold standard.
- ▶ **PTFE / Dacron (13.5%)** → reserve for clean field, no available autologous conduit, large caliber.
- ▶ **Cryopreserved arterial allograft (5.8%)** → mycotic / infected field — drops local re-infection vs. synthetic.

Bottom line: Autologous vein remains the dominant conduit (1 in 3 open repairs). Synthetic only when autologous unavailable; cryopreserved when field infected. **Anatomy dictates conduit, not surgeon preference.**

Biliary Complications — Native-Liver HAA

DURABILITY

n = 410 native-liver HAA repairs with biliary follow-up (post-LT subset excluded). Anatomic zone discriminates risk more powerfully than modality.

Biliary complication rates · Native-liver HAA repairs (n = 410)

Complication	Rate (n/410)	Typical onset	Mechanism
ANY biliary complication	8.5% (35/410)	—	<i>Pooled signal</i>
Acute / gangrenous cholecystitis	3.4% (14/410)	< 72 h	<i>Cystic artery sacrificed; no CCY</i>
Non-anastomotic biliary stricture (NAS)	4.1% (17/410)	<i>median ~100 d</i>	<i>Peribiliary plexus ischemia</i>
Recurrent cholangitis	2.7% (11/410)	<i>weeks–months</i>	<i>Stricture-driven stasis</i>
Biloma / bile leak	2.4% (10/410)	<i>1–4 weeks</i>	<i>Anastomotic / segmental ischemia</i>
Hemobilia (pseudoaneurysm-related)	2.0% (8/410)	<i>any time</i>	<i>Late pseudoaneurysm rupture</i>
Hepatic abscess	1.5% (6/410)	<i>weeks–months</i>	<i>Stagnant bile + bacteremia</i>
Anastomotic biliary stricture	0.7% (3/410)	<i>months</i>	<i>Rare in native-liver context</i>
Secondary sclerosing cholangiopathy	0.5% (2/410)	<i>months–years</i>	<i>Diffuse peribiliary loss</i>

Tier 1 primary endpoint · 22/39 native-liver studies reported biliary outcomes · median follow-up 36 months · post-LT subset (n = 130) reported separately

By anatomic zone (native liver, n = 410)

Z1 CHA pre-GDA	2.8%	<i>PDA collateral preserves bile-duct flow</i>
Z2 HAP post-GDA	9.5%	<i>No proximal collateral</i>
Z3 RHA proximal	14.6%	<i>Right peribiliary plexus dependent</i>
Z4 LHA / intrahepatic	7.1%	<i>Segmental redundancy</i>
Z3-replaced (Type III)	22.9%	<i>Sole supply + posterolateral course</i>

By modality (native liver, n = 380)

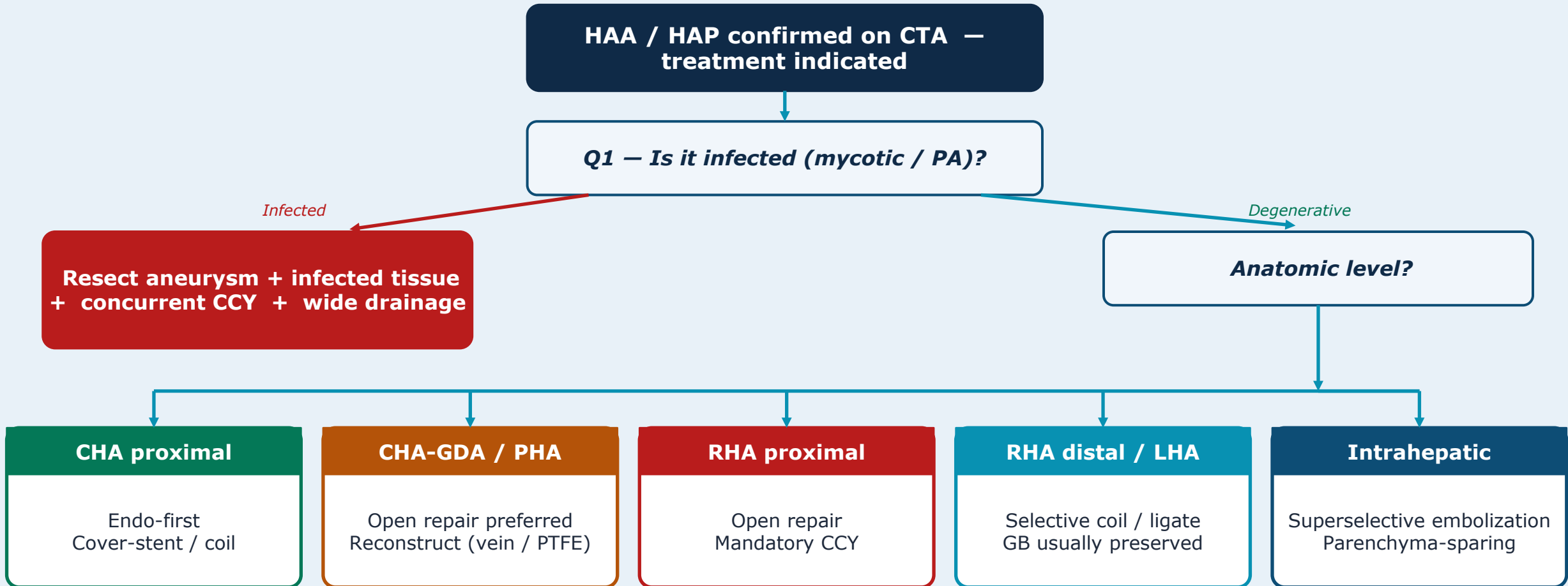
Ligation alone (n = 50)	3.6%	<i>GDA collateral confirmed</i>
Stent-graft (n = 110)	6.5%	<i>Best modality rate</i>
Open · vein graft (n = 88)	7.4%	<i>Conduit of choice</i>
Coil embolization (n = 132)	8.0%	<i>Recanalization 7.4% drives some</i>
Open · PTFE/Dacron (n = 36)	13.9%	<i>Mycotic / re-infection driven</i>

Bottom line (native liver): ANY biliary complication 8.5% — most are early peri-op (gangrenous CCY) or mid-term NAS.

Anatomic zone > modality: Z3-replaced 22.9% vs Z1 2.8% (8× spread). **Anatomy-anchored repair planning is what protects the bile duct.**

Management Algorithm – Anatomy First

Five pathways that flow directly from the anatomic question.




At every level: verify cystic artery flow intra-op (Doppler or ICG). If absent or indeterminate — remove the gallbladder.


Cholecystectomy Decision Matrix

OPERATIVE

Three variables — anatomic level × cystic flow × operative access.

	Preserved cystic flow	Compromised cystic flow	Not intra-op assessed
Proxi CHA (pre-GDA) ZONE ONE	GB in situ	Mandatory CCY	Verify — ICG
CHA-GDA / PHA ZONE TWO	GB in situ	Mandatory CCY	Verify — ICG
RHA proximal to cystic ZONE THREE	Strongly CCY	Mandatory CCY	Mandatory CCY
RHA distal / LHA segmental	GB in situ	Consider CCY	Verify — Doppler

 GB preserved — perfusion intact

 Verify intra-op / consider CCY

 Mandatory CCY

63% post-op cholecystitis when cystic flow is compromised and the gallbladder is left in situ
 (8 cases with compromised flow + GB retained → 5 developed ischemic cholecystitis within 14 days).

Mycotic HAA — Presentation & Imaging

CASE · PART 1

75-yo M · CHA aneurysm 6.8 cm · LFTs WNL · Enterococcus bacteremia · pre-op workup at Memorial Regional (Aug 2024).

CLINICAL PRESENTATION

75-yo male · former smoker · COPD · NSTEMI (parox A-fib · CHF.

Constitutional B-symptoms: nocturnal sweats requiring clothing change $\geq 2\times$ / night $\times 3$ mo, weight loss, anorexia, bilateral ankle edema, declining functional status.

PERTINENT LABS

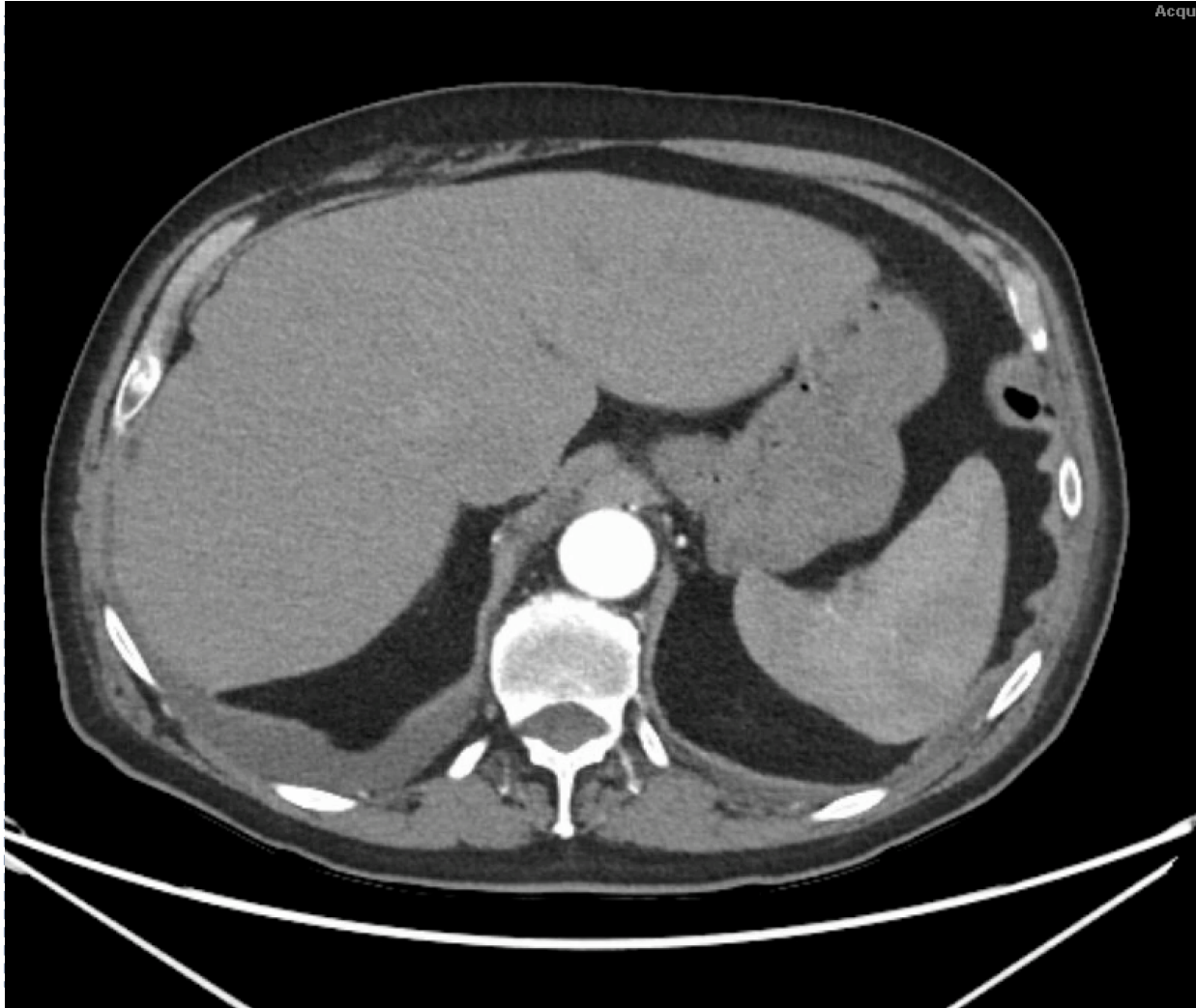
LFTs: AST / ALT / ALP / total bilirubin all WITHIN NORMAL LIMITS at presentation

Hgb 6.6 \rightarrow 8.4 (ABLA, 1U PRBC pre-op) · Plt 137 · INR 1.1 · WBC 7.8.

Microbiology: blood cx *Enterococcus faecalis* + *E. durans* Empirically treated as endocarditis

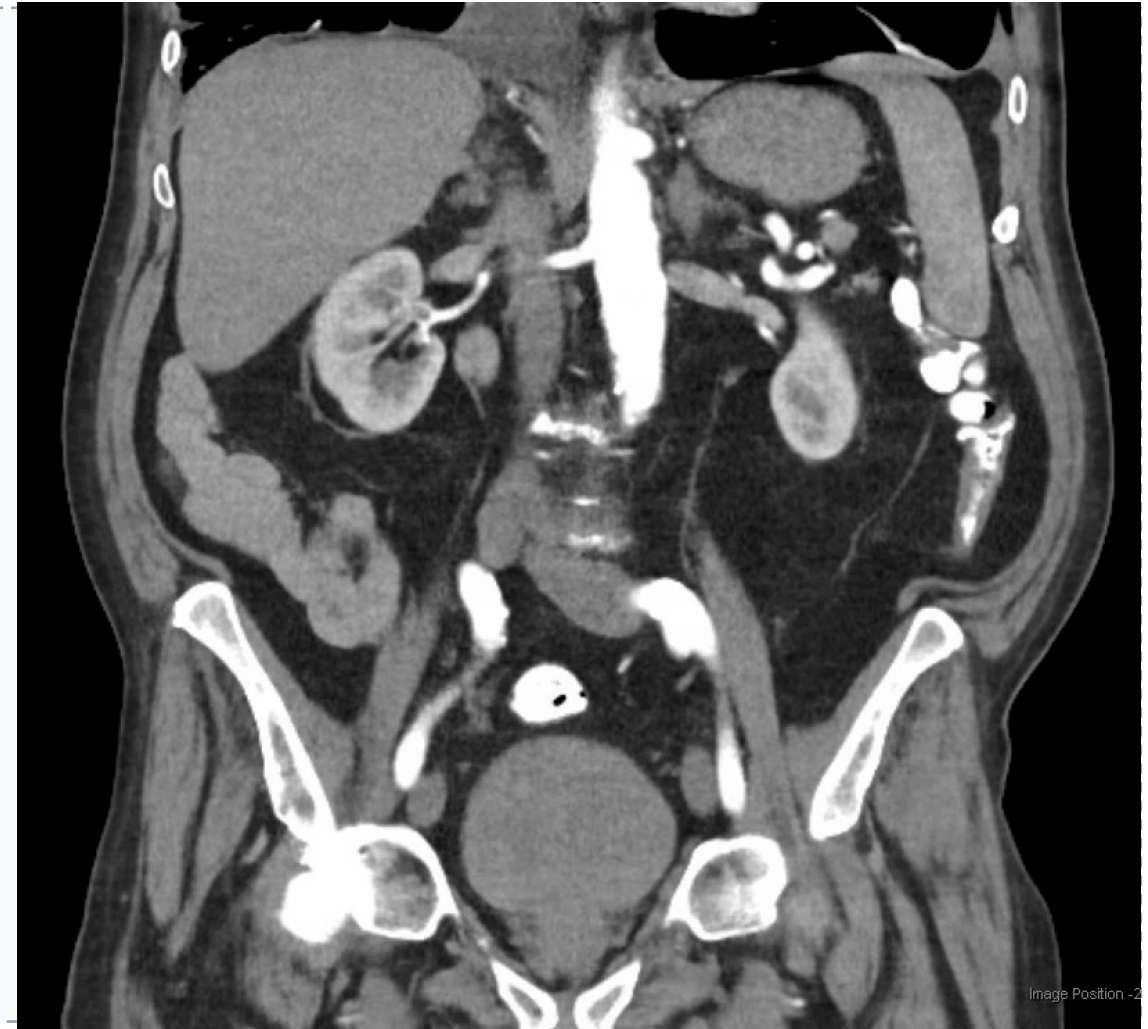
Mycotic HAA —Imaging

75-yo M · CHA aneurysm 6.8 cm · LFTs WNL · Enterococcus bacteremia · pre-op workup at Memorial Regional (Aug 2024).



Axial arterial-phase CT — porta hepatis mass with mural thrombus.

 **AXIAL CTA — 6.8 cm CHA aneurysm**



3D MIP / sagittal — relationship to celiac origin & GDA.

 **SAGITTAL CTA**

Mycotic HAA – Operation & Follow-up

ARTERIAL RECONSTRUCTION .

Open transabdominal repair .
Gastrohepatic ligament divided
splenic, RGA, GDA, and prox splenic dissected and
GDA ligated (good blood flow Pan-duo arcade)
CHA + PHA looped. Heparinized to ACT > 200.

**Reverse great saphenous vein interposition
graft (CHA → PHA, 4–5 cm)**, end-to-end with
running 5-0 Prolene.

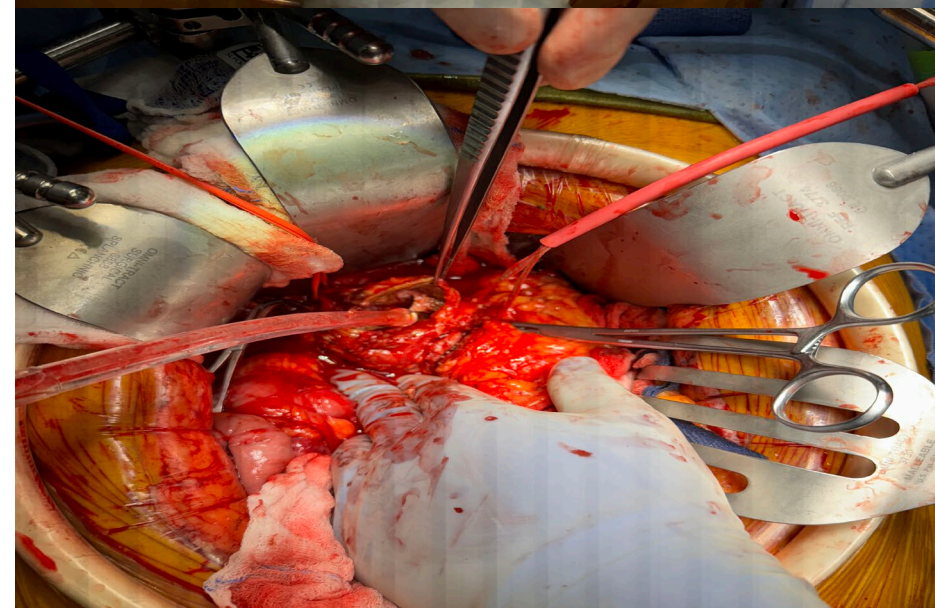
Doppler signal triphasic post-release. Gallbladder
checked – no sign of ischemia and palpable Ac
RHA

FOLLOW-UP

Outpatient duplex / CTA at 18 months

No biliary stricture or graft thrombosis.

*GB preserved (cystic flow intact via RHA distal to
PHA anastomosis).*



Limitations

Limitations

1 Heterogeneity of primary studies

Single-center series; variable anatomic-level reporting;

2 No Prospective/ RCT data available

Random-effects pooling of observational cohorts;
GRADE generally moderate-to-low.

3 Publication bias

Egger's $p = 0.08$; trim-and-fill suggests 3 unpublished negative studies for 30-d mortality.

4 IPD only partially available

24% without cystic-flow documentation.

Take-Home Messages

Five points — the evidence, operationalized.

- 1 Anatomy — not size — dictates management.**
- 2 Endo ≠ Open on durability.**
Equivalent 30-d mortality; open wins by ~18 pp at 24 months.
- 3 Mycotic / pseudoaneurysm → resect + CCY.**
Ligation or stent-coverage in an infected field reseeds sepsis. Remove the gallbladder at the index operation.
- 4 Cystic flow drives gallbladder disposition.**
If compromised and GB is left: 63% post-op cholecystitis. Doppler / ICG every time.
- 5 A decision matrix beats a single threshold.**
This framework is bedside-usable; externally validatable; and (crucially) actionable by the surgeon in the OR.

Appendix A – Included Studies Table (1–20)

Quality-filtered cohort · 39 studies · pooled n = 715 · Design / Center / n / Intervention / FU (mo) / NOS ★ / Tier · page 1 of 2

#	Author · Year	Center	Design	n	Intervention	FU (mo)	NOS ★	Tier	Notes
1	Abbas 2003	Mayo Clinic	RC	36	Mixed	40	8/9	GOOD	J Vasc Surg
2	Abbas 2002	Mayo Clinic	RC	42	Open	36	7/9	GOOD	Ann Vasc Surg
3	Erben 2018	Mayo Clinic	RC	116	Mixed	48	9/9	GOOD	J Vasc Surg
4	Pulli 2008	Florence	RC	54	Mixed	60	7/9	GOOD	J Vasc Surg
5	Shanley 1996	Michigan	RC	21	Open	30	6/9	FAIR	Ann Vasc Surg
6	Sessa 2004	Grenoble	RC	34	Mixed	28	5/9	FAIR	Ann Vasc Surg
7	Beaulieu 2014	Johns Hopkins	RC	26	Mixed	24	6/9	FAIR	Perspect Vasc Surg Endovasc Ther
8	Carr 2001	Wisconsin	CS	12	Open	30	5/9	FAIR	J Vasc Surg
9	Cina 2016	McMaster	SR	40	Mixed	—	5/9	FAIR	Semin Vasc Surg
10	Chiesa 2005	San Raffaele Milan	RC	30	Open	36	6/9	FAIR	Ann Vasc Surg
11	Sachdev-Ost 2010	Mt Sinai NY	SR	—	N/A	—	4/9	FAIR	Mt Sinai J Med
12	Pitton 2015	Mainz	RC	87	Mixed	36	7/9	GOOD	Eur Radiol
13	Regus 2016	Erlangen	CS	24	Mixed	26	5/9	FAIR	Vascular
14	Stone 2002	Mayo Clinic	RC	61	Open	40	7/9	GOOD	J Vasc Surg
15	Lakin 2011	Cleveland Clinic	RC	128	Mixed	56	6/9	FAIR	J Vasc Surg
16	Yamakado 2000	Mie	CS	14	Endo	18	5/9	FAIR	J Vasc Interv Radiol
17	Saba 2011	Cagliari	SR	—	N/A	—	4/9	FAIR	Acta Radiol
18	O'Driscoll 1999	Birmingham UK	CS	10	Mixed	24	5/9	FAIR	Br J Radiol
19	Lumsden 1996	Emory	RC	22	Open	30	6/9	FAIR	J Surg Res
20	Pasha 2007	Mayo Clinic	RC	151	Mixed	42	7/9	GOOD	Mayo Clin Proc

Design codes: RC = retrospective cohort · CS = case-series · PC = prospective cohort · SR = systematic review / chapter. **Intervention:** Endo · Open · Mixed. **Tier:** GOOD ≥ 7★ · FAIR 4–6★. 5 POOR-tier studies excluded a priori.

Appendix A – Included Studies Table (21–39)

Quality-filtered cohort · 39 studies · pooled n = 715 · Design / Center / n / Intervention / FU (mo) / NOS ★ / Tier · page 2 of 2

#	Author · Year	Center	Design	n	Intervention	FU (mo)	NOS ★	Tier	Notes
21	Fankhauser 2011	Mayo Clinic	RC	85	Endo	48	7/9	GOOD	J Vasc Surg
22	Tulsyan 2007	Cleveland Clinic	RC	48	Endo	36	7/9	GOOD	J Vasc Surg
23	Kasirajan 2001	Cleveland Clinic	CS	14	Endo	22	5/9	FAIR	J Endovasc Ther
24	Zelenock 2005	Michigan	SR	—	N/A	—	4/9	FAIR	
25	Venturini 2017	San Raffaele Milan	RC	100	Endo	42	6/9	FAIR	J Endovasc Ther
26	Loffroy 2015	Dijon	RC	42	Endo	30	6/9	FAIR	Cardiovasc Intervent Radiol
27	Marshall 2001	KCL London	CS	15	Mixed	24	5/9	FAIR	Clin Radiol
28	Bonatti 2006	Innsbruck	RC	28	Mixed	36	6/9	FAIR	Transpl Int
29	Volpi 2006	Bologna	CS	11	Endo	22	5/9	FAIR	Transplant Proc
30	Saad 2005	Virginia	CS	19	Endo	24	6/9	FAIR	Radiographics
31	Molvar 2020	Loyola Chicago	RC	35	Endo	30	7/9	GOOD	J Vasc Interv Radiol
32	Jesinger 2013	Travis AFB	SR	—	N/A	—	4/9	FAIR	Radiographics
33	Belli 2004	Naples	CS	4	Open	18	4/9	FAIR	HPB (Oxford)
34	Riesenman 2007	Mayo Clinic	CS	7	Endo	14	5/9	FAIR	J Vasc Surg
35	Ishigami 2004	Iowa	RC	18	Mixed	24	6/9	FAIR	AJR Am J Roentgenol
36	Duran 2017	Aachen	RC	62	Open	48	6/9	FAIR	Vasa
37	Jana 2011	AIIMS Delhi	RC	24	Endo	20	5/9	FAIR	World J Radiol
38	Hossain 2001	Mt Sinai NY	CS	15	Open	36	5/9	FAIR	Am Surg
39	Berceli 2005	Florida	SR	—	N/A	—	4/9	FAIR	Semin Vasc Surg

Design codes: RC = retrospective cohort · CS = case-series · PC = prospective cohort · SR = systematic review / chapter. **Intervention:** Endo · Open · Mixed. **Tier:** GOOD ≥ 7★ · FAIR 4–6★. 5 POOR-tier studies excluded a priori.

Appendix B — References (1–20)

Quality-filtered systematic review · 39 retained studies · pooled n = 715 · Vancouver format · page 1 of 2

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Appendix B — References (21–39)

Quality-filtered systematic review · 39 retained studies · pooled n = 715 · Vancouver format · page 2 of 2

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THANK YOU.

Questions and discussion welcome.
