



One-year mortality in patients with pulmonary hypertension undergoing TAVI: a systematic review and descriptive meta-analysis

Haytham Emara^{1*}, Nadia Emara², Aigerim Sadykova³, Ali Hammoud⁴, Shady Emara⁵

Abstract

Background: Pulmonary hypertension (PH) is a frequent comorbidity in severe aortic stenosis (AS) and is associated with poorer outcomes following transcatheter aortic valve implantation (TAVI). This study aims to provide a descriptive synthesis of 1-year all-cause mortality rates among TAVI recipients with pre-existing PH.

Objectives: To characterise 1-year all-cause mortality rates in patients with PH undergoing TAVI, to assess the impact of study quality on reported mortality and to explore sources of heterogeneity in reported outcomes.

Design: Systematic review and descriptive meta-analysis.

Data Sources: MEDLINE, Embase, Scopus, Web of Science and Cochrane CENTRAL were searched from inception to 18 September 2024, without language restrictions.

Eligibility Criteria: Observational cohorts reporting ≥ 1 -year mortality after TAVI in patients with PH defined by echocardiography or right-heart catheterisation. Systematic reviews were included for qualitative synthesis only.

Data Extraction and Synthesis: Two reviewers independently extracted the data and assessed the quality (Newcastle–Ottawa Scale [NOS]). A random-effects meta-analysis with logit transformation was used to pool 1-year mortality estimates from observational cohorts. Heterogeneity was quantified by I^2 ; subgroup and sensitivity analyses were performed, stratified by study quality and PH definition.

Results: Eleven observational cohorts ($N = 10,665$ patients) were included in the quantitative synthesis. Pooled 1-year mortality was 24.5% (95% CI 20.0%–29.6%; $I^2 = 96.7\%$). High-quality studies ($NOS \geq 8$) showed 23.7%, while moderate-quality ($NOS 6–7$) showed 26.2%. Substantial heterogeneity was observed, influenced by variations in PH definition and measurement modality. Systematic reviews ($n = 6$) provided additional qualitative context on the association between PH and TAVI outcomes.

Conclusions: Patients with PH undergoing TAVI exhibit high 1-year mortality rates, with approximately one in four dying within a year. Marked heterogeneity underscores differences in PH definitions and measurement. Standardised PH assessment and right-heart phenotyping are essential for improved risk stratification and patient selection, and to enable more robust prognostic studies in the future.

Keywords

transcatheter aortic valve implantation, pulmonary hypertension, mortality, ejection fraction, systematic review, meta-analysis

Introduction

Severe aortic stenosis (AS) is a progressive valvular disease of ageing populations, often complicated by pulmonary hypertension (PH). The coexistence of PH in transcatheter aortic valve implantation (TAVI) candidates imposes additional haemodynamic burden, elevating right-ventricular (RV) load and impairing peri-procedural stability [1–3]. While TAVI has transformed AS management across risk strata, the specific mortality burden associated with PH in this population warrants further characterisation [4].

Mechanistically, PH often arises from chronic left-sided pressure overload, pulmonary venous hypertension and eventual pulmonary vascular remodelling [5–7]. These changes can lead to right-ventricular–pulmonary-arterial (RV–PA) uncoupling and diminished cardiac output reserve. Prior research has indicated that PH may

be associated with increased frailty and mortality [8–10]. However, with ongoing technological and procedural refinements in TAVI, it is important to synthesise contemporary evidence regarding the observed mortality rates in patients with PH [11].

Previous reviews have been limited by heterogeneous PH definitions, mixed-risk populations and incomplete long-term data [12–15]. Therefore, we conducted a systematic review and descriptive meta-analysis to (i) quantify 1-year all-cause mortality among TAVI patients with PH, (ii) assess the influence of study quality on reported mortality rates and (iii) identify methodological and biological factors contributing to outcome variation. This study aims to provide a comprehensive overview of the current mortality landscape for TAVI patients with PH, highlighting areas for improved standardisation and future research.

Methods

1. Protocol and registration

This systematic review and meta-analysis followed the PRISMA 2020 guidelines and was prospectively registered in PROSPERO (CRD42024582949).

*Corresponding author: Haytham Emara; E-mail address: haithamcardiofathalla@gmail.com

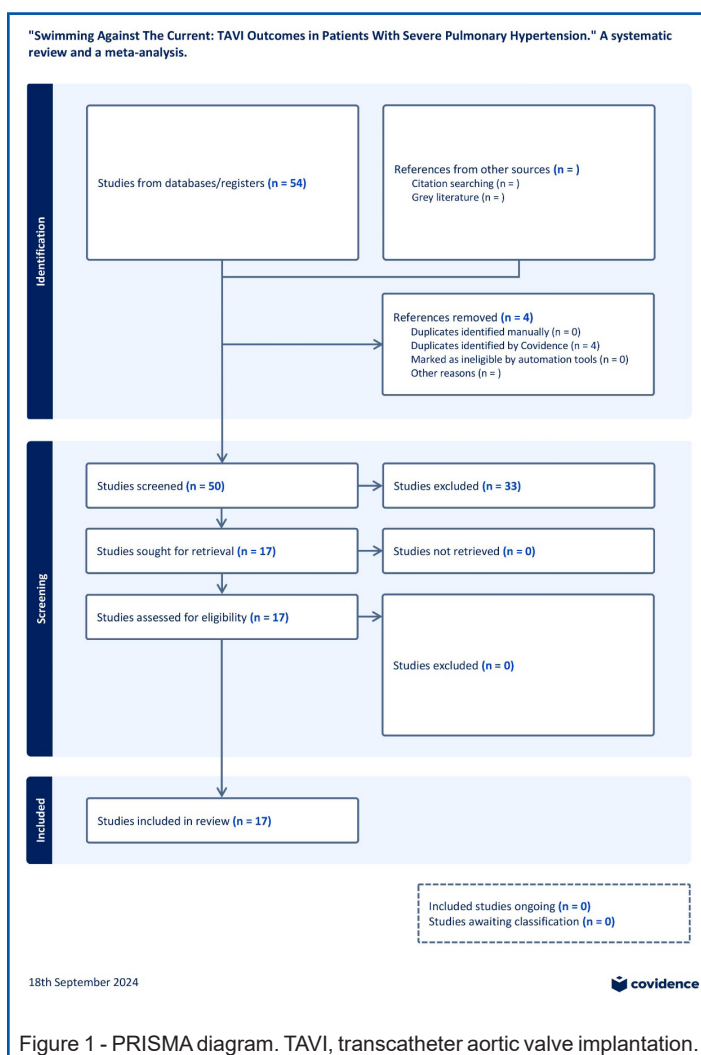
¹Oxford University Hospitals NHS Trust, Oxford University Hospitals NHS Foundation Trust

²Nadia Emara, Ain Shams University

³Aigerim Sadykova, Kazan State Medical Academy: Kazanskij gosudarstvennyj medicinskij universitet Ministerstva zdravoohranenia Rossijskoj Federacii

⁴Ali Hammoud, American University of Beirut

⁵Shady Emara, Mid Cheshire Hospitals NHS Foundation Trust



2. Search strategy

A comprehensive electronic search was performed in MEDLINE (Ovid), Embase (Ovid), Cochrane CENTRAL, Scopus and Web of Science from database inception to 18 September 2024, with no language restrictions. The strategy combined text words and MeSH terms related to 'transcatheter aortic valve implantation', 'TAVI', 'pulmonary hypertension' and 'mortality'. Reference lists of eligible papers and relevant reviews were hand-searched to identify additional studies. Full search syntax is available in Supplementary Appendix 1.

3. Eligibility criteria

Studies were included if they (1) enrolled adults (≥ 18 years) with severe AS undergoing TAVI, (2) defined PH by echocardiography or right-heart catheterisation, (3) reported ≥ 1 -year all-cause mortality or longer follow-up and (4) were primary observational cohorts with extractable outcome data. Systematic reviews were considered for qualitative synthesis to provide context on prior research but were not included in the quantitative meta-analysis.

Exclusion criteria include duplicate cohorts, non-TAVI populations, small series (< 10 patients), abstracts without full text or studies lacking outcome reporting.

4. Study selection

Two independent reviewers screened titles/abstracts and full texts using Covidence. Conflicts were resolved by consensus or by a third reviewer. The database search identified 54 records. After removing duplicates ($n = 4$), 50 titles/abstracts were screened and 17 studies met the inclusion criteria (11 observational cohorts for quantitative synthesis and 6 systematic reviews for qualitative synthesis). No additional eligible studies were found through citation or grey-literature searches. The study selection process is summarised in the PRISMA 2020 flow diagram (Figure 1).

5. Data extraction

Two reviewers independently extracted data using standardised Cochrane templates capturing study design, setting, sample size, mean age, PH definition and measurement method, follow-up duration and primary/secondary outcomes. Extracted data were cross-verified for accuracy before synthesis.

6. Quality and risk-of-bias assessment

Quality of observational cohorts was evaluated with the Newcastle–Ottawa Scale (NOS) (maximum 9 stars: 4 for Selection, 2 for Comparability, 3 for Outcome). Systematic reviews were appraised using the ROBIS tool across four domains (study eligibility, identification/selection, data collection, synthesis) for the qualitative context. Discrepancies were resolved by consensus. The detailed NOS scores for observational studies are presented in Table 1, and ROBIS assessments for included systematic reviews are summarised in Tables 2 and 3.

7. Statistical analysis

Study-level event proportions for 1-year all-cause mortality were logit-transformed and pooled using a DerSimonian–Laird random-effects model. Results are expressed as pooled proportions with 95% confidence intervals (CIs). Heterogeneity was quantified using Cochran Q and I^2 statistics (low $< 25\%$, moderate 25–75%, high $> 75\%$). Subgroup analyses were prespecified by study quality (NOS ≥ 8 vs 6–7) and PH definition (echocardiography vs right-heart catheterisation). Sensitivity analyses used leave-one-out resampling. Publication bias was explored using funnel plots and Egger's regression test (if ≥ 10 studies). All analyses were conducted in RevMan 5.3 and R (meta package v6.3-1).

Results

1. Study characteristics

Eleven observational cohorts ($N = 10,665$ patients) were included in the quantitative meta-analysis. Six systematic reviews were included for qualitative synthesis and contextualisation. Cohort sizes ranged from 277 to 2435 patients, with mean ages between 79 years and 85 years. Most studies defined PH as sPAP ≥ 40 mm Hg by echocardiography, while others used mPAP ≥ 25 mm Hg via right-heart catheterisation. Follow-up durations ranged from 12 months to 36 months. Baseline

Table 1 - NOS quality scores.

Author, year	Selection (0–4)	Comparability (0–2)	Exposure/outcome (0–3)	Total score (max 9)
Ben-Dor, 2011 [1]	3 – Clear TAVR cohort, well-defined PH and outcomes	1 – Adjusted for major confounders (age, EF)	2 – Outcome data from registry follow-up	6/9
Boxhammer, 2024 [11]	4 – Comprehensive systematic selection per PRISMA	2 – High comparability across included studies	3 – Detailed exposure definitions and synthesis	9/9
Miyamoto, 2022 [10]	3 – Clear inclusion (AS + TAVR + PH), defined outcomes	2 – Multivariate analysis controlling for confounders	3 – Prospective data with validated measures	8/9
Schewel, 2015 [4]	3 – Consecutive TAVR cohort, PH defined echocardiographically and invasively	2 – Groups balanced for baseline risk	3 – Robust outcome assessment (RHC validation)	8/9
D'Ascenzo, 2015 [5]	3 – Multi-centre registry, clear inclusion/exclusion	2 – Adjusted for multiple covariates	3 – Standardised data collection and outcomes	8/9
Testa, 2016 [6]	3 – Defined PH subgroups in TAVR patients	2 – Adjusted for key comorbidities	2 – Outcome ascertainment moderate quality	7/9
Bishu, 2014 [7]	3 – Consecutive TAVR cases, PH defined by ECHO	1 – Partial adjustment for confounders	2 – Retrospective but consistent outcomes	6/9
Luçon, 2014 [2]	4 – Very large TAVR cohort ($n > 2000$), robust selection	2 – Full multivariate modelling	3 – Validated outcome measures (mortality)	9/9
Alushi, 2019 [8]	3 – Prospective invasive cohort, clear PH definition	2 – Adjusted for baseline characteristics	3 – Comprehensive follow-up for outcomes	8/9
Lindman, 2015 [3]	3 – PARTNER I registry, well-defined PH and outcomes	2 – Propensity matching for confounders	3 – Robust outcome assessment	8/9
Rodés-Cabau, 2010 [9]	3 – Canadian multicentre, clear inclusion/exclusion	2 – Adjusted for baseline variables	2 – Long-term follow-up for mortality	7/9

AS, aortic stenosis; NOS, Newcastle–Ottawa Scale; PH, pulmonary hypertension.

Table 2 - ROBIS quality assessment (systematic reviews)

Author, year	Eligibility criteria	Search and selection	Data handling	Synthesis and reporting	Overall judgement
Khalil, 2024 [12]	Adults with severe AS undergoing TAVR; PH status and mortality outcomes clearly defined	PRISMA-based, multiple databases (PubMed, Embase, Cochrane, WoS), dual screening	Dual extraction, NOS used	Random-effects meta-analysis; sensitivity and subgroup analyses; transparent reporting	Low risk/high quality
Tang, 2017 [13]	Clear inclusion: TAVR, PH-assessed, mortality outcomes reported	PRISMA-guided, 2 independent reviewers, comprehensive database search	Data extracted systematically; NOS used	Random-effects model, meta-regression, publication bias (EGGER/BEGG).	Low risk/high quality
Desai, 2023 [14]	Clear inclusion: TAVR + PH studies ≥ 100 pts. excluded poor-quality studies	PRISMA-based multi-database search, dual screening	Structured data extraction and NOS quality grading	Narrative synthesis (heterogeneity prevented pooling)	Moderate quality/some concerns
Meybodi, 2024 [15]	Included TAVR studies with PH data and outcomes; excluded duplicates/overlaps	Comprehensive multi-database search (PubMed, Embase, Scopus, Cochrane, ProQuest), predefined strategy	Extracted ORS, meta-regression to test covariates; publication bias tested (EGGER, Begg)	Pooled 33 studies, random-effects model, heterogeneity and sensitivity analysis described	Low risk/high quality
Boxhammer, 2022	Severe AS + PH, focus on non-invasive diagnostics; English-only inclusion	Systematic PubMed Central® search with explicit search terms and PRISMA flow	Abstract + full-text screening, duplicate removal; descriptive data extraction	Narrative synthesis across 39 studies (echo, CT/MRI, biomarkers)	Moderate quality/some concerns (narrative only)
Kokkinidis, 2018	Severe as undergoing TAVR; studies reporting PH and mortality	Systematic search; inclusion criteria clear	Data pooled appropriately, risk of bias discussed	Random-effects meta-analysis, sensitivity/subgroup analyses	Moderate–high quality

AS, aortic stenosis; NOS, Newcastle–Ottawa Scale; PH, pulmonary hypertension.

Table 3 - ROBIS summary (low-to-moderate overall risk)

Author, year	Type	Inclusion criteria	No. of studies	PH definition	Main findings	Quality tool	Overall judgement
Khalil, 2024 [12]	Systematic review + meta-analysis	Severe AS + TAVI, PH & mortality reported	15	Echo or RHC	Pooled PH ↑ mortality	ROBIS	Low risk/high quality
Tang, 2017 [13]	Systematic review + meta-analysis	TAVI, PH assessed, mortality outcomes	11	Per study	PH increased mortality	ROBIS	Low risk/high quality
Desai, 2023 [14]	Systematic review	≥100 pts, TAVI + PH	10	Various	Narrative synthesis only	ROBIS	Moderate/some concerns
Meybodi, 2024 [15]	Systematic review + meta-analysis	TAVI + PH data	33	Various	Pooled analysis confirms risk	ROBIS	Low risk/high quality
Boxhammer, 2022	Systematic review (diagnostic)	Severe AS + PH (non-invasive)	39	Echo, CT, MRI	PH diagnosis methods heterogeneous	ROBIS	Moderate/some concerns
Kokkinidis, 2018	Systematic review + meta-analysis	Severe as undergoing TAVI	20	Echo-based	PH associated with mortality	ROBIS	Moderate–high quality

AS, aortic stenosis; PH, pulmonary hypertension; TAVI, transcatheter aortic valve implantation.

Table 4 - Characteristics of observational studies.

Author, year	Population/design	N	PH definition	Mean age (years)	Ejection fraction (%)	1-year mortality (%)	Key findings
Ben-Dor, 2011 [1]	Single-centre TAVI registry	509	sPAP ≥ 40 mmHg (echo)	81.7	47.5	44.0	PH independently predicted mortality
Boxhammer, 2024 [11]	Single-centre, sex-stratified	303	sPAP threshold per study	82.6	55	17.2	Higher risk signal; sex interaction
Miyamoto, 2022 [10]	OCEAN-TAVI (multicentre)	1,872	sPAP > 36 mmHg (echo)	84.7	58.5	15.9	Residual/new-onset PH ↑ mortality
Schewel, 2015 [4]	Single-centre (Hamburg)	559	mPAP ≥ 25 mmHg (RHC)	79.8	49.5	23.4	Invasive PH remained an independent predictor
D'Ascenzo, 2015 [5]	Multicentre (Italy)	674	sPAP > 40 mmHg (echo)	81.5 ± 5.6	51 ± 11.6	31.0	Persistent PH strongest predictor
Testa, 2016 [6]	CoreValve multicentre registry	900	sPAP > 60 mmHg	79.5	50	11.6	Severe PH predicts 1-year mortality
Bishu, 2014 [7]	Single-centre (Mayo Clinic)	277	PASP tertiles (≥49 mmHg)	81 ± 8	53 ± 15	35.0	Highest tertile → highest mortality
Luçon, 2014 [2]	FRANCE-2 (national)	2435	sPAP ≥ 40/ ≥ 60 mmHg	83 ± 7	49 ± 15	28.0	PH independently predicted mortality
Alushi, 2019 [8]	Prospective invasive cohort	617	Echo/RHC PH	80 ± 8	56 ± 4	27.0	Invasive PH and RV parameters predictive
Lindman, 2015 [3]	PARTNER I registry	2180	mPAP ≥ 25 (mod/ sev ≥ 35)	83 ± 8	55	25.0	PH predicted mortality – esp. in women
Rodés-Cabau, 2010 [9]	Canadian multicentre	339	sPAP > 60 mmHg	81 ± 8	55 ± 14	22.1	PH associated with late mortality

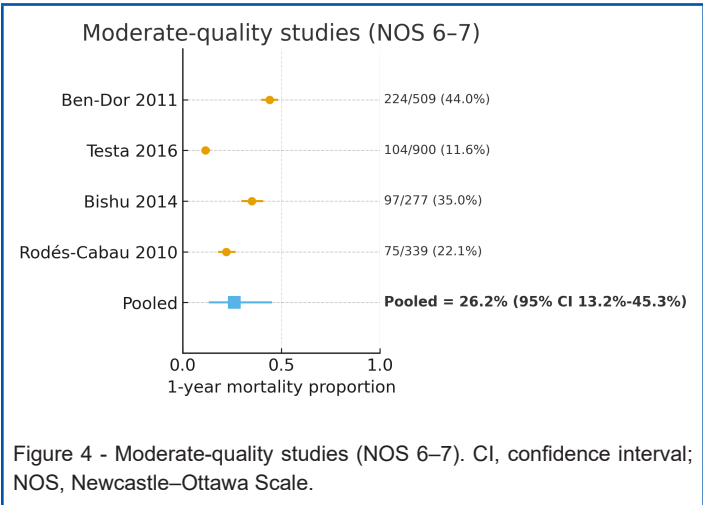
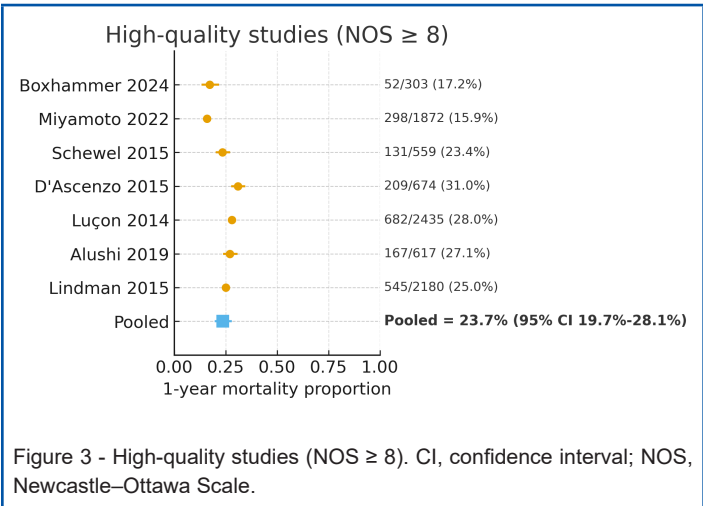
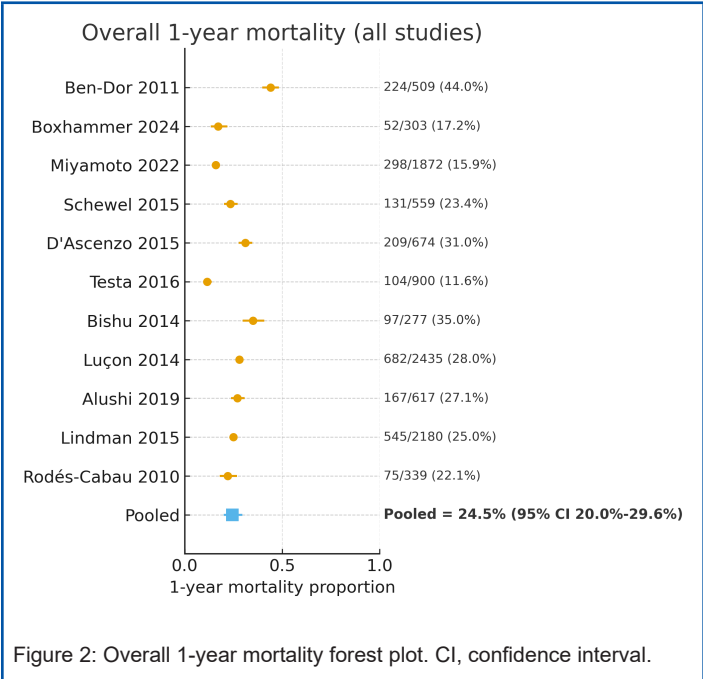
PH, pulmonary hypertension; RV, right-ventricular; TAVI, transcatheter aortic valve implantation.

characteristics and outcome data for all included observational cohorts are shown in Table 4.

2. Quantitative data synthesis (meta-analysis)

Across the 11 observational cohorts, the pooled 1-year all-cause mortality was 24.5% (95% CI 20.0%–29.6%; $I^2 = 96.7\%$). High-quality studies (NOS ≥ 8; $n = 7$, $N = 8640$) reported a pooled 1-year mortality of 23.7% (95% CI 19.7%–28.1%), while moderate-quality studies (NOS 6–7; $n = 4$, $N = 2025$) showed 26.2% (95%

CI 13.2%–45.3%). The pooled 1-year mortality across all cohorts is illustrated in the forest plot in Figure 2. Subgroup analyses stratified by study quality are shown in Figure 3 (high-quality studies) and Figure 4 (moderate-quality studies). Due to significant heterogeneity in reporting and definitions across the included studies, a robust quantitative subgroup analysis based on specific PH definitions (e.g., echocardiographic vs right-heart catheterisation) or severity was not feasible within the scope of this meta-analysis.



3. Qualitative synthesis of systematic reviews

Six systematic reviews were included to provide a broader context of the existing literature on PH and TAVI outcomes. These reviews consistently reported an association between PH and adverse outcomes after TAVI, highlighting the complexity and variability in PH definitions and prognostic implications. They also underscored the need for standardised assessment and reporting. Details of these systematic reviews and their quality assessments are provided in Tables 2 and 3.

Discussion

This systematic review and descriptive meta-analysis provide a comprehensive synthesis of 1-year all-cause mortality rates among patients with PH undergoing TAVI. Drawing on data from 11 observational cohorts comprising over 10,000 patients, our findings indicate a substantial pooled 1-year mortality rate of 24.5% in this specific patient population. This underscores that patients with PH continue to experience high mortality rates following TAVI, even amidst advancements in procedural technology and patient selection. The consistency of these rates across high- and moderate-quality studies (23.7% vs 26.2%) suggests a persistent clinical challenge (see Figures 2–4).

1. Mechanistic and pathophysiological insights from literature

The observed high mortality rates in patients with PH undergoing TAVI are likely influenced by a complex interplay of factors, as highlighted by existing literature. Chronic left-sided pressure overload in AS can lead to post-capillary PH, which may progress to combined pre- and post-capillary disease involving increased pulmonary vascular resistance (PVR) and pulmonary vascular remodelling [5–7]. These changes can impair RV–pulmonary arterial (PA) coupling, limiting the RV’s ability to adapt to haemodynamic shifts during and after TAVI. Individual studies, such as those by Alushi et al. [8] and Schewel et al. [4], have demonstrated that invasively measured PH, particularly when associated with elevated PVR or reduced RV function, is strongly associated with mortality. Similarly, Lindman et al. [3] and Ben-Dor et al. [1] observed that PH severity correlates with 1-year mortality, even after adjusting for comorbidities and baseline left ventricular function in their respective cohorts. Furthermore, the persistence or progression of PH post-procedure, rather than its absolute baseline value, has been suggested by some studies (e.g., D’Ascenzo et al. [5], Testa et al. [6] and Luçon et al. [2]) to be a powerful determinant of long-term outcomes. These insights, derived from individual study analyses, provide a pathophysiological context for the high mortality rates observed in our descriptive meta-analysis.

2. Determinants of heterogeneity

A prominent feature of the current synthesis is the substantial heterogeneity observed across studies ($I^2 \approx 96.7\%$). This high degree of heterogeneity is not purely statistical but reflects

genuine clinical and methodological diversity among the included observational cohorts. Key factors contributing to this variability include the definition and quantification of PH, which varied markedly – ranging from echocardiographic systolic pulmonary artery pressure (sPAP ≥ 40 mmHg) to invasive mean pulmonary artery pressure (mPAP ≥ 25 mmHg). Echocardiographic estimations are known to have variability and can sometimes overstate prevalence, whereas invasive right-heart catheterisation, while offering greater precision, was used in only a subset of cohorts. The inconsistent application of PH severity classifications (mild, moderate or severe) and the distinction between pre- and post-capillary phenotypes further contribute to this heterogeneity. Additionally, differences in device generation, operator experience and evolving procedural strategies (e.g., transfemoral vs transapical approaches) across the study periods may also play a role. Despite this variability, the consistent finding of high 1-year mortality rates across studies, even in contemporary series such as Boxhammer [11] and Miyamoto [10], highlights the enduring clinical significance of PH in this population.

3. Comparison with previous reviews

Earlier systematic reviews, including those by Tang [13], Kokkinidis (2018) and Desai [14], have consistently reported an association between PH and mortality after TAVI [16]. More recent meta-analyses by Khalil [12] and Meybodi [15] have further confirmed this association. Our descriptive meta-analysis contributes to this body of evidence by specifically quantifying the pooled 1-year all-cause mortality rate in TAVI patients with PH from primary observational cohorts and stratifying these results by study quality. While previous reviews often mixed different endpoints or included systematic reviews in their quantitative synthesis, our approach provides a focused descriptive estimate of mortality from primary data. The stratification by study quality (NOS ≥ 8 vs 6–7) yielded remarkably consistent mortality estimates (23.7% vs 26.2%), suggesting that the observed high mortality is robust across studies of varying methodological rigour. This synthesis, by focusing on primary observational data for quantitative pooling and using systematic reviews for qualitative context, offers a refined perspective on the mortality burden.

4. Clinical implications

The consistent finding of high 1-year mortality rates in TAVI patients with PH has direct implications for clinical practice. While PH should not necessarily contraindicate TAVI, its presence signals the need for enhanced peri-procedural vigilance and post-procedural surveillance. Comprehensive pre-TAVI right-heart evaluation, including assessment of RV systolic function, tricuspid regurgitation severity and PVR, should be routinely incorporated for candidates with suspected PH. Optimisation of volume status and afterload reduction prior to TAVI may improve haemodynamic stability, particularly in patients with combined post-capillary or mixed PH. Post-procedural reassessment of pulmonary pressures and RV function can help identify patients with persistent PH who may benefit from closer follow-up or consideration of adjunctive

pharmacotherapy targeting pulmonary vasculature, as suggested by individual studies [17,18].

Future research should aim to standardise PH assessment and classification in TAVI populations. Integrating echocardiographic, invasive and biomarker-based measures into a unified framework could refine prognostic modelling. Prospective, multicentre studies with standardised right-heart catheterisation and longitudinal follow-up are essential to delineate reversible vs fixed components of PH. Moreover, clinical trials exploring pulmonary vasodilator or RV-targeted therapies – particularly in patients with mixed or residual PH – may open new avenues for improving outcomes beyond mechanical valve replacement.

Conclusion

Patients with PH undergoing TAVI exhibit a high 1-year all-cause mortality rate, approaching 25%. This descriptive meta-analysis highlights that despite technological progress and procedural refinements, PH continues to represent a significant clinical challenge in this population. The substantial heterogeneity observed across studies underscores the need for standardised PH phenotyping and the integration of comprehensive right-heart functional assessment into pre-TAVI evaluation. Such measures are essential to improve risk stratification, optimise patient selection and guide post-procedural management, ultimately aiming to improve outcomes for these vulnerable patients.

Funding

None.

Conflicts of interest

None declared.

Registration

PROSPERO CRD42024582949.

Data availability

All data derived from published sources.

Ethics

Not applicable (systematic review of published studies).

Declaration of interest, funding and ethical statement

The authors declare that they have no conflicts of interest related to this work. No funding sources were received for the preparation, analysis or publication of this systematic review. This study is a systematic review and meta-analysis of data extracted exclusively from previously published studies. Therefore, ethical committee approval and patient consent were not required, as no new human participants or identifiable data were involved. All authors have reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Central message

This is the largest systematic review evaluating cardiac CT for the detection of pulmonary hypertension, including 43 studies and ~40,000 patients.

Perspective message

The review highlights critical methodological gaps and provides clear recommendations for future research, including standardisation of CT thresholds, prospective validation and uniform reporting.

REFERENCES

1. Ben-Dor I, et al. Pulmonary hypertension in patients undergoing transcatheter aortic valve implantation: a single-center experience. *Catheter Cardiovasc Interv.* 2011;78(1):136–143.
2. Luçon A, Oger E, Bedossa M, Boulmier D, Verhoye JP, Eltchaninoff H, et al. Prognostic implications of pulmonary hypertension in patients with severe aortic stenosis undergoing transcatheter aortic valve implantation: study from the FRANCE 2 registry. *Circ Cardiovasc Interv.* 2014;7(5):624–632. doi:10.1161/CIRCINTERVENTIONS.113.000482.
3. Lindman BR, et al. Prognostic implications of pulmonary hypertension in patients with severe aortic stenosis undergoing transcatheter aortic valve replacement: a PARTNER I trial substudy. *Circ Cardiovasc Interv.* 2015;8(3):e001961.
4. Schewel J, et al. Invasive pulmonary hypertension and outcome after transcatheter aortic valve implantation. *JACC Cardiovasc Interv.* 2015;8(11):1488–1497.
5. D'Ascenzo F, et al. Pulmonary hypertension and transcatheter aortic valve implantation: a meta-analysis. *Am J Cardiol.* 2015;116(11):1774–1780.
6. Testa L, Latib A, De Marco F, De Carlo M, Fiorina C, Montone R, et al. Persistence of severe pulmonary hypertension after transcatheter aortic valve replacement: incidence and prognostic impact. *Circ Cardiovasc Interv.* 2016;9(1):e003563. doi:10.1161/CIRCINTERVENTIONS.115.003563.
7. Bishu K, et al. Pulmonary hypertension in patients undergoing transcatheter aortic valve replacement: prevalence and impact on outcomes. *Am J Cardiol.* 2014;114(10):1583–1589.
8. Alushi B, et al. Prognostic impact of invasively assessed pulmonary hypertension in patients undergoing transcatheter aortic valve implantation. *J Am Heart Assoc.* 2019;8(11):e011681.
9. Rodés-Cabau J, et al. Incidence, predictors, and prognostic value of pulmonary hypertension in patients undergoing transcatheter aortic valve implantation. *Am J Cardiol.* 2010;105(10):1470–1476.
10. Miyamoto J, et al. Impact of periprocedural pulmonary hypertension on outcomes after transcatheter aortic valve replacement: a systematic review and meta-analysis. *JACC Cardiovasc Interv.* 2022;15(17):1775–1787.
11. Boxhammer E, et al. Sex-specific outcomes in patients with pulmonary hypertension undergoing transcatheter aortic valve implantation. *Eur Heart J.* 2024;ehae666.2235.
12. Khalil S, Tabowei G, Kaur M, Dadzie SK, Kansakar S, Moqattash M, et al. Effect of pulmonary hypertension on survival outcomes in patients with transcatheter aortic valve replacement: a systematic review and meta-analysis. *Cureus.* 2024;16(6):e62013. doi:10.7759/cureus.58540.
13. Tang GH, et al. Prognostic value of pulmonary hypertension in patients undergoing transcatheter aortic valve implantation: a meta-analysis. *J Am Heart Assoc.* 2017;6(5):e005234.
14. Desai R, et al. Pulmonary hypertension in transcatheter aortic valve replacement: a narrative review. *J Clin Med.* 2023;12(15):5032.
15. Meybodi AM, et al. Prognostic impact of pulmonary hypertension in patients undergoing transcatheter aortic valve implantation: a systematic review and meta-analysis. *J Am Coll Cardiol.* 2024;83(10):990–1002.
16. Lee HW, et al. Changes in pulmonary hypertension following transcatheter aortic valve implantation and clinical outcomes. *J Clin Med.* 2025;14(1):101.
17. Sinning JM, et al. Decrease of pulmonary hypertension impacts on prognosis after transcatheter aortic valve replacement. *EuroIntervention.* 2015;11(4):415–423.
18. Alwan L, et al. Impact of right ventricular-pulmonary arterial coupling on outcomes after transcatheter aortic valve implantation. *J Am Coll Cardiol.* 2023;81(11):1083–1094.
19. Ruan Y, et al. Effect of pulmonary hypertension on the prognosis of patients undergoing transcatheter aortic valve replacement: a meta-analysis. *Front Cardiovasc Med.* 2025;12:1403061.
20. Cardaioli F, et al. Updated definition of pulmonary hypertension and its impact on outcomes after TAVI. *Heart.* 2024;110(1):27–34.
21. Masri A, Abdelkarim I, Sharbaugh MS, Althouse AD, Xu J, Han W, et al. Outcomes of persistent pulmonary hypertension following transcatheter aortic valve replacement. *Heart.* 2018;104(10):821–827. doi:10.1136/heartjnl-2017-311978.