Acute Decompensated Aortic Stenosis: State of the Art Review

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Abstract

Aortic stenosis (AS) is a progressive disease that carries a poor prognosis. Patients are managed conservatively until satisfying an indication for transcatheter aortic valve implantation (TAVI) or surgical aortic valve replacement (SAVR) based on AS severity and the presence of symptoms or adverse impact on the myocardium. Up to 1 in 3 TAVIs are performed for patients with acute symptoms of dyspnoea at rest, angina, and/or syncope - termed acute decompensated aortic stenosis (ADAS) and require urgent aortic valve replacement. These patients have longer hospital length of stay, undergo physical deconditioning, have a higher rate of acute kidney injury and mortality compared to stable patients with less severe symptoms. There is an urgent need to prevent ADAS and to deliver pathways to manage and improve ADAS-related outcomes. We provide here a contemporary review on epidemiological and pathophysiological aspects of ADAS, with a focus on the impact of ADAS from clinical and economic perspectives. We will offer also a global overview of the available evidence for treatment of ADAS and with priorities suggested for addressing current gaps in the literature and unmet clinical needs to improve outcomes for AS patients.

Keywords: aortic stenosis, decompensated aortic stenosis, transcatheter aortic valve implantation, surgical aortic valve replacement, risk stratification, cardiogenic shock,

Definition of acute decompensated aortic stenosis

Aortic stenosis (AS) is one of the most prevalent valvular heart diseases, affecting 1.3% of people >65 years old, with 0.7% having either moderate or severe disease (1). The prevalence increases with age, with severe AS affecting up to 3.4% of people >75 years old (2). It is a progressive condition that eventually results in the development of symptoms and heart failure and increases the risk of mortality. Medical therapy has not yet proven to slow down or treat AS; treatment requires aortic valve replacement (AVR). Indications for AVR have expanded over time, however, many patients do not receive a timely AVR and present with acute decompensated aortic stenosis (ADAS).

Although no universally established definition exists, ADAS has broadly been described in the literature as the need for hospitalisation secondary to severe AS and is primarily governed by the severity and onset of symptoms. Patients are New York Heart Association (NYHA) IV, Canadian Cardiovascular Society (CCS) IV (in the absence of epicardial coronary artery disease) or have had syncope. Some clinicians consider symptoms on minimal exertion to fulfil the criteria for ADAS. These symptoms develop over a short period, and patients can often pinpoint their onset. Patients either report an acute deterioration in symptoms or the onset of new symptoms. This is in contrast, for example, to progressive dyspnoea that eventually leads to breathlessness at rest. Both require urgent aortic valve replacement, but the latter is often associated with fewer comorbidities and better outcomes. From a pathological perspective, patients have evidence of congestion and poor organ perfusion (3). We propose the following definition for ADAS: in patients with severe AS, the development of new symptoms or acute deterioration in existing symptoms over the past week that result in either NYHA IV, CCS IV (in the absence of epicardial coronary artery disease) or syncope.

At the extreme end of ADAS are patients in AS-induced cardiogenic shock. These patients are critically unwell and often need life-saving emergent AVR or balloon aortic valvuloplasty (BAV)(4,5).

Impact of acute decompensated aortic stenosis

Mortality associated with acute decompensated aortic stenosis

Conservative management of ADAS portends a dismal prognosis with a mortality of 30.5% at 1 year (3). This is worse among conservatively managed patients in cardiogenic shock, 30-day mortality is reported at 46% (5). AVR improves outcomes in ADAS. However, compared to non-ADAS patients, there remains an increased risk of short, mid and long-term mortality (3,6–15). A meta-analysis demonstrated a hazard ratio (HR) of death whilst in hospital of 2.09, 95% confidence interval (CI): 1.39-3.14, at 30 days post-TAVI of 2.29, 95% CI: 1.69-3.10 and at 1-year post-TAVI of 1.96, 95% CI: 1.55-2.49 (16).

Several factors have been identified as predictors of mortality among patients with ADAS (Table 1). Pre-existing patient-related factors are similar to non-ADAS patients and include chronic obstructive pulmonary disease, atrial fibrillation, chronic kidney disease, frailty, low body mass index and higher surgical risk (7,8,11,13,17). Greater severity of extra-aortic valve dysfunction or damage quantified using a well validated echocardiographic classification system (18) does impact mortality in ADAS patients at 1 year post-TAVI (17).

Procedural factors are also important, with patients requiring non-transfemoral TAVI access, cardiopulmonary bypass, who develop acute kidney injury (AKI) and stroke are at increased risk of mortality (8,9,11). It is yet to be determined whether improving any of these prognostic factors pre- or post-intervention can improve outcomes in ADAS.

Morbidity and procedural complications associated with acute decompensated aortic stenosis

TAVI procedural complications are largely similar between ADAS and non-ADAS patients except for AKI and bleeding. The definition of AKI varies between studies- however, many have documented an increased rate of AKI among ADAS patients compared to stable patients (6,8–10,19,20), with a meta-analysis reporting a HR of 2.48, 95% CI: 1.85-3.32 (16). The mechanism for AKI is likely to be multifactorial: reduced cardiac output may impair renal perfusion and the use of iodinated contrast for computed tomography, invasive coronary angiography, and TAVI within a short window of time may increase the risk of contrast induced nephropathy. Ultimately, more prevalent comorbidities (such as atrial fibrillation) and worse renal function at baseline may increase the risk of further deterioration. Accordingly, the rate of dialysis among ADAS compared with non-ADAS is increased (HR: 2.37, 95% CI: 1.95-2.88) (16). SAVR often impacts renal function to a greater extent than TAVI. Thus in the context of ADAS, treatment with SAVR needs to be carefully considered before proceeding (14). Some studies have identified an increased risk of bleeding in ADAS (HR: 1.62, 95% CI:1.27-2.08) (16) and subsequently need for blood transfusions (10). This is especially true when ADAS is treated with SAVR (14). Reported rates of vascular complications, pacemaker implantations or strokes between ADAS and non-ADAS patients are similar with TAVI (6).

ADAS patients have less improvement in symptoms and left ventricular ejection fraction (LVEF) at 30-days and 1-year post-TAVI than non-ADAS patients (10,19). Even after ADAS is treated with aortic valve replacement, patients continue to have an increased risk of heart failure hospitalisation (3). This suggests that certain myocardial characteristics, such as focal fibrosis, may be irreversible or deteriorate post-valve replacement (21,22), whilst some comorbidities such as AF may persist (23).

Social and economic implications of acute decompensated aortic stenosis

Hospital length of stay (LoS) has been universally longer for ADAS patients compared to non-ADAS patients (8–10,14,15,19) with means of 11-31 days (6,9,14,15). ADAS patients also tend to have an increased need for further care in rehabilitation or nursing facilities (6,8). Unsurprisingly, SAVR patients tend to have longer LoS than TAVI patients (14). Longer LoS directly correlates with increased healthcare costs. ADAS patients also require more time spent in intensive care, and ventilatory, cardiac and renal support, adding significantly to costs (5,6,8,24). Longer length of stay and admission to critical care is associated with physical deconditioning and functional dependence. This is especially debilitating for frail or elderly patients and has repercussions on post-hospital care, quality of life and independence (25). Of note, among non-ADAS patients, several studies based on UK, Italian and USA-healthcare systems have demonstrated the cost-effectiveness of TAVI against medical therapy and SAVR in inoperable, high, intermediate and low risk patients (26–28). Studies have not reported costs of treating ADAS patients, however, this is likely to be greater than non-ADAS patients. Demonstrating cost-effectiveness of various treatment strategies in ADAS will be important to guide treatment pathways.

Epidemiology of acute decompensated aortic stenosis

Among patients undergoing TAVI, the prevalence of ADAS ranges between 6.6 and 34.5% (3,6,8–10,13,15,19,29,30). This is likely to be higher as many elective TAVIs are performed in patients who have had previous hospitalisation for AS-related decompensation and patients eventually managed medically are often not included in ADAS studies (19). Among the SAVR, the prevalence is reported at 11.4% (14). A smaller proportion of approximately 3.5% present with ADAS and cardiogenic shock (4). Worryingly, ADAS appears to be increasing, with several countries reporting an increasing proportion of TAVIs being performed for ADAS (6,29). An ageing population, may account for some of this

increase (31). The COVID pandemic has had an impact on normal clinical practice and although speculative, may account for some of this increase.

Natural history of aortic stenosis

AS increases the afterload of the left ventricle (LV), increasing wall stress and reducing cardiac output. The LV myocardium compensates by undergoing hypertrophy (32,33) and adopting distinct patterns of remodelling (34). Sex and several comorbidities that frequently coexist with AS such as diabetes, hypertension, obesity and chronic kidney disease contribute to the development of left ventricular hypertrophy (33,35–38). Simultaneously there is an accumulation of focal and diffuse fibrosis (39) with a transmural gradient (endocardium more than the epicardium) (40). Patients will often develop diastolic dysfunction (41) and eventually many will go on to develop systolic dysfunction (42). As the severity of AS increases, most patients will eventually become symptomatic. Historical studies have shown that mortality increases significantly at this point (43). Symptoms in AS correlate with markers of diastolic (E/e' and left atrial volume index) and systolic function (cardiac output and stroke volume index) (44). In severe AS, aortic valve haemodynamics do not seem to determine the presence or severity of symptoms, highlighting the impact of myocardial structure and function as determinants of symptom onset and prognosis (44).

<u>Dyspnoea</u>

Diastolic dysfunction correlates with dyspnoea, specifically E/e' and left atrial volume indexed - both markers of left ventricular filling pressures (44). Pathological changes described above: left ventricular hypertrophy, myocardial fibrosis and slowed early active relaxation because of abnormal calcium handling affect diastolic function and together contribute towards the development of dyspnoea (45,46). Cardiac remodelling especially

collagen architecture and the degree of hypertrophy in relation to wall stress alters left ventricular systolic function (47,48), which in turn may influence dyspnoea. However, thus far, markers of LV systolic function have not demonstrated a correlation with dyspnoea (44,49).

Syncope

Syncope occurs from hypotension resulting in transient cerebral hypoperfusion. These patients may have lower E/e' and LV mass. Consequently, they have lower stroke volume indexed and cardiac output compared with those with angina or dyspnoea. Low diastolic filling pressures limit LV volumes and cardiac output, whilst low LV mass may be inadequate to maintain sufficient stroke volume for the degree of AS, rendering patients susceptible to cerebral hypoperfusion (44,45). In patients with severe AS, syncope usually occurs during exercise, when stroke volume fails to adapt coupled with abnormal vascular responses and reduced total peripheral resistance (49,50).

Angina

Coronary haemodynamics are altered in AS, such that myocardial oxygen supply may not meet increases in oxygen demand, rendering the myocardium susceptible to ischaemia (51). Demand increases due to left ventricular hypertrophy and wall stress- both a consequence of increased afterload. Coronary supply is restricted due to several factors: capillary rarefaction- capillary network does not expand with myocardial hypertrophy, endocardial compression due to increased end-diastolic pressure, myocardial fibrosis which increases perfusion distances, reduced diastolic perfusion time- as more time is spent ejecting blood from the LV in systole and reduced coronary flow reserve- vascular dilatation at rest to increase blood supply to meet the heart's increased demand, albeit constraining further

increases in supply during exercise (40,45,52–54). Anaemia may also contribute to angina and is commonly associated with AS (55). Angina can also be the consequence of epicardial coronary stenosis and therefore angiography, whether invasive or with computed tomography, is required to differentiate between this and AS-related demand-supply mismatch. Traditional markers of ischaemia, such as chest pain, troponin elevations and ECG changes cannot reliably differentiate between a type 1 and type 2 myocardial infarction (56).

Pathophysiology of acute decompensated aortic stenosis

Given the differences in cardiac structure and function that drive each symptom, the pathophysiology of ADAS differs depending on the predominant symptom. The most frequent symptom is dyspnoea at rest, followed by syncope (10,11,19). Dyspnoea is often secondary to acute heart failure or decompensation of chronic heart failure (57). Comorbidities also contribute to the risk of ADAS and in turn influence prognosis. Arrhythmias, in particular atrial fibrillation (AF) may trigger acute decompensation. Although AF is prevalent among ADAS patients, studies have not differentiated between new onset and chronic AF (8,9,11,12).

Baseline characteristics of acute decompensated aortic stenosis

Compared to non-ADAS patients, those with ADAS often have a higher prevalence and severity of comorbidities. Chronic kidney disease (eGFR<60ml/kg/1.7m^2), left ventricular systolic dysfunction (LVEF<50-55%), AF, pulmonary diseases (especially chronic obstructive pulmonary disease), coronary artery disease (either a history of revascularisation or untreated significant stenosis) and frailty (Rockwood clinical frailty score>5) are more common in ADAS. Patients are at higher surgical risk, tend to have poorer renal function and lower haemoglobin levels (3,8–12,14,15,17,19,29).

From an echocardiographic perspective, studies have reported a higher prevalence of coexisting significant valvular heart disease (3) in particular aortic regurgitation and failed bioprosthetic valves among ADAS patients (8). Higher pulmonary artery systolic pressures have also been reported (9). Concomitant myocardial disease such as cardiac amyloidosis may increase the risk of ADAS (58).

ADAS was more prevalent among ethnic minorities in a large study (8)- a cohort known to have limited access to healthcare in many health care systems (59). Undiagnosed valvular heart disease was found to be twice as high among the two most deprived socioeconomic quintiles compared to the most affluent quintile (1). This suggests that limited access to healthcare and cultural differences in the perception of health may be crucial factors leading to ADAS.

Patients in cardiogenic shock tend to have even higher surgical risk (STS score 23.4 ± 11.6) and greater severity of cardiac dysfunction (mean LVEF 30 ± 14%, AVA 0.61 ± 0.17, PASP 54.1 ± 11.5) (5). Similar findings were reported by another study (20).

These studies have demonstrated that at the time of decompensation, patients with ADAS have a higher prevalence of comorbidities and more cardiac dysfunction compared to non-ADAS.

Predictors of acute decompensated aortic stenosis

Predicting which patients will decompensate is crucial to guide timely valve intervention and to prevent ADAS. Based on the studies discussed above, patients with multiple comorbidities, who are frail and have greater cardiac dysfunction will be at greater risk of ADAS. Additionally, novel markers of myocardial dysfunction or adverse cardiac remodelling may predict ADAS. They represent subclinical indicators of myocardial decompensation.

Fibrosis can be quantified using cardiovascular magnetic resonance (CMR) imaging and computed tomography (60,61). Diffuse fibrosis can be tracked using quantification of extracellular volume (ECV). Greater ECV, expressed as a percentage of myocardial volume, is associated with more symptomatic patients (NYHA III/IV), cardiac remodelling (greater LV mass, left atrial volumes) and a reduction in LVEF and right ventricular ejection fraction. (62).

Coronary haemodynamics (epicardial and microvascular) are affected in AS, such that myocardial oxygen demand increases and supply reduces (56). Myocardial perfusion reserve (MPR), an index of the myocardium's ability to increase blood flow during stress compared to rest, can be measured using CMR. In asymptomatic moderate-severe AS patients followed for 374 (351-498) days, MPR was moderately associated with symptom onset (area under the receiver operating curve: 0.56) (63).

Strain imaging is a sensitive indicator of cardiac function (64). Patients with AS demonstrate progressively worsening strain over time, in particular- global longitudinal strain (GLS) in the subendocardial layer (65). Worse GLS is associated with the development of symptoms in patients with asymptomatic severe AS (66).

Brain natriuretic peptide (BNP) and N-terminal proBNP (NT-proBNP) have been extensively evaluated in patients with AS (67). BNP levels correlate well with symptom onset and using different cut-offs, studies have demonstrated AUC between 0.84-0.86 (68,69).

Diffuse fibrosis, microcirculatory dysfunction, reduced GLS and elevated BNP have all demonstrated diagnostic ability to predict the onset of symptoms. They could prove valuable in predicting ADAS. It is likely that a multiparametric approach will be required rather than a single test. Further studies are needed to evaluate their role in predicting ADAS.

Treatment options for acute decompensated aortic stenosis

Balloon Aortic Valvuloplasty (BAV)

Historically, BAV was commonly used in patients with decompensated AS (70,71). It has the advantage over TAVI of being a shorter procedure, using less contrast and smaller sheath sizes (8Fr vs 14-16Fr) (72,73). It can also act as a bridge-to-decision in some patients where it is unknown whether an aortic valve replacement would be beneficial and where patient evaluation under more stable clinical conditions is required. In critically unwell patients where a short procedure to relieve afterload and improve haemodynamics is required, BAV is an attractive option (72). Additionally, in an emergency when a pre-TAVI CT scan to guide valvular sizing and vascular access is not possible, BAV may be a safer alternative as it would reduce the risk of vascular complications related to large sheaths and incorrectly sized valves. BAV does result in symptom improvement for the majority of patients (61% of survivors at 2 years) (71) so it can also be used as a palliative measure (7). However, the haemodynamic change is marginal, such that many patients continue to have severe AS: post-procedure, aortic valve area increases from 0.61 ± 0.2 to 0.8 ± 0.2cm2; $p<0.001$, whilst peak AV gradient reduces from 87 ± 22 to 66 ± 22mmHg; $p<0.001$ (7). Outcomes of BAV without subsequent aortic valve replacement remain poor (7)- 30-day mortality of 46.2% (95% CI: 30.3-62.5%). Rehospitalisation was frequent: 64%, rates of moderate to severe aortic regurgitation were high: 15% at 6 months and restenosis was fairly quick: aortic valve area reduced from 0.78 ± 0.31cm^2 at baseline to 0.68 ± 0.25cm^2 at 6 months post-BAV (71). Patients often present with acute heart failure and require definitive intervention with AVR (14).

ADAS patients in cardiogenic shock have poor outcomes with BAV- at 1 year 75% were either dead or had recurrent cardiogenic shock (5,24). These outcomes among the sickest cohort of ADAS patients illustrate the ineffectiveness of BAV in this patient cohort. First line therapy using TAVI appears to be more effective than BAV, even if the latter is

considered as a bridge to aortic valve replacement. Therefore, if feasible, TAVI should be the preferred option over BAV.

Transcatheter aortic valve implantation

Procedural success in ADAS is high albeit lower than non-ADAS. One study reported lower device success among patients with ADAS (85.2 vs 91.8%; p=0.03) and higher use of a second valve (11.1 vs 4.4%; p=0.05), however CT was only used in a quarter of patients (19). A meta-analysis did demonstrate a statistically lower device success among ADAS patients, albeit a small numerical difference between ADAS and non-ADAS (92.6 vs 93.7; p=0.007) (74). Procedural complications among ADAS patients are similar to non-ADAS patients with the exception of AKI (9,19), bleeding and dialysis which tend to be higher among ADAS patients (74). Differences in practice regarding the use of pre-procedural CT planning, annular pre-dilatation, rapid pacing, post-dilatation and the definitions used for procedural and device success account for the heterogeneity in the reported literature. TAVI has also been used in patients with cardiogenic shock. One study demonstrated successful device implantation but unsurprisingly poor overall outcomes with a 30 day mortality of 33.3% (20). A meta-analysis of such patients identified a 30 day mortality of 22.6% (95% CI: 12-35.2%) (75). TAVI as a first-line strategy is proving to be the optimum treatment strategy for patients with ADAS.

Surgical aortic valve replacement

A few studies have evaluated the role of SAVR in ADAS. A national registry was evaluated to compare SAVR with TAVI among propensity matched patients with AS and acute heart failure. Survival did not differ at 30 days, 1 and 2 years post-AVR. SAVR patients did have longer in-hospital length of stay, AKI, bleeding and transfusion rates

compared to TAVI patients (14). Compared to elective SAVR, patients with acute heart failure undergoing SAVR had higher rates of bleeding, transfusion, re-sternotomy, mechanical support, AKI and hospital LoS (14). A small non-randomised study compared SAVR to TAVI in patients with cardiogenic shock. SAVR patients had a lower surgical risk score, higher blood transfusion rates and no difference in mortality up to 1 year compared to TAVI (76). Additionally, TAVI can offer larger relative orifice areas and is associated with a lower risk of patient prosthesis mismatch- both important considerations especially in patients with impaired LVEF (77). Concomitant treatment of coronary artery disease may be required in addition to AVR. Proposals to guide the choice and timing of such treatment are discussed elsewhere (51).

Current challenges in the management of aortic stenosis

AS is now more treatable than ever with the advent of TAVI and improvements in SAVR, and peri and post-interventional care. Timing of valve replacement is critical to balance the potential risks of intervention with the benefits of a new valve. Although recent progress has been made to optimise this (78,79), many patients do not fulfil guideline-directed indications for aortic valve replacement (80). These patients are monitored at regular intervals using serial echocardiograms and clinical evaluations ('watchful-wait' approach) until an indication is reached (81). However, this approach has several flaws: first, undiagnosed valvular heart disease is very common (affecting 1 in 2 adults ≥65 years) (13). Second, assessing symptoms can be challenging as patients may not sufficiently exert themselves to illicit symptoms or may not recognise their symptoms and thus be wrongly labelled as asymptomatic (82). Third, severity of AS can also be challenging to establish with up to a third of patients having discordant echocardiographic findings (83). Lastly, the rate of progression of AS is variable and can be rapid, up to a reduction in aortic valve area of 0.5

cm^2/year (84). Consequently, a significant proportion of ADAS patients are already known to have pre-existing severe AS- between 40-57% (9,19). Whilst among those referred for TAVI evaluation, 0.5% die before having their TAVI (19). Adopting a 'watchful wait' approach can result in sudden cardiac death among asymptomatic patients- 0.39% in mild to moderate AS to 1.8% in severe AS (78,85). This highlights the importance of predicting ADAS, timely AVR and lowering the existing threshold for AVR.

Potential gaps and proposed strategies towards improving outcomes in ADAS

Screening for aortic stenosis

It is estimated that 1.1-1.3% of patients over 65 years have undiagnosed AS with 0.7% demonstrating severe AS (1,86). Among those aged>75 years the prevalence of moderate-severe AS is 2.6% (87). A significant proportion of AS patients are not treated (88) and many present with ADAS. A new diagnosis of severe AS was identified in 17.6% of patients presenting with ADAS (9). Cardiac auscultation can identify patients for further evaluation with Doppler echocardiography (86). Alternatively using echocardiography as a screening tool can identify, with greater accuracy, valvular heart disease and heart failure. Two echo based screening studies identified undiagnosed valvular heart disease in 36-51% of participants (1,89). However, these screening services present a significant financial and workforce commitment, which may be cost-effective if particular populations such as the elderly are targeted along with abbreviated or targeted scanning protocols to minimise time and expertise requirement. Artificial intelligence may have a potential role in supporting image acquisition and analysis, thus enabling a wider group of staff with minimal expertise and training to perform such screening strategies sustainably. Artificial intelligence applied to ECG analysis has demonstrated good accuracy for identifying AS in a large population (area

under the receiver operating curve (AUC) 0.85) and may present an alternative screening opportunity (90).

Prevention of acute decompensated aortic stenosis

By determining the rate of progression of both AS and associated myocardial remodelling, the interplay between AS and comorbidities and identifying which phenotypes are at highest risk of decompensation, patients can be risk stratified and treatment expedited for those at highest risk.

Optimising the timing and reducing waiting times for valve replacement will be key to reducing the incidence of ADAS. Patient initiated follow-up is a simple method many healthcare organisations employ to empower patients and expedite investigations and treatment at the onset of a patient's symptoms (91). There is evidence that patient initiated follow-up can improve outcomes in some populations (92), with AS patients, especially those who are asymptomatic potentially benefitting from such schemes.

Lower thresholds for AVR in patients with AS using better risk stratification will prevent ADAS. Identifying these high-risk cohorts will require a better understanding of the mechanisms and predictors of ADAS. Prospective studies that prove the benefits of AVR compared to conservative management are required to support earlier treatment. Several trials are ongoing in patients with moderate AS with reduced LVEF, symptoms or cardiac damage (93–95).

Timing of aortic valve replacement for ADAS

Several studies have demonstrated a signal indicating a survival advantage among ADAS patients treated early. Among patients in cardiogenic shock, BAV within 48 hours of starting inotropic support or the diagnosis of cardiogenic shock was associated with a lower

mortality at 1 year compared to >48 hours (59 vs 90%; p=0.01) (5,96). Among patients without cardiogenic shock, two retrospective observational studies on ADAS patients found that delayed treatment with TAVI was associated with an increased mortality rate at 1 and 2 years (11,12). Earlier treatment has theoretical advantages by reducing the time spent with left ventricular outflow obstruction and its associated adverse impact on the left ventricle, tissue hypoperfusion- especially the kidneys, and reducing the risk of malignant arrhythmias and heart failure. Prospective studies evaluating the impact of time to treatment in ADAS are needed.

Conclusions

ADAS is common and impacts mortality, morbidity, and healthcare services. TAVI is likely to be the optimal therapeutic option for ADAS. Screening for AS, optimising timing to aortic valve replacement for AS and expediting treatment once decompensated may improve outcomes.

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Graphical abstract legend

Acute decompensated aortic stenosis (ADAS) is characterised by the acute onset or deterioration of symptoms related to aortic stenosis. It is common and impacts mortality, morbidity and healthcare provision. Several clinical factors can predict ADAS, whilst several others are prognostically important. Screening for AS, optimising timing to aortic valve replacement for AS and expediting treatment once decompensated may improve outcomes. Vector image were obtained from vecteezy.com