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The Critical Role of Nurses in managing patients with Valvular heart disease

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Abstract

Valvular heart disease (VHD) poses a significant global health burden, with increasing prevalence due to an aging population. Aortic stenosis (AoS), the most common form of VHD, is characterized by progressive narrowing of the aortic valve, leading to poor prognosis if left untreated. Aortic valve replacement, either surgical or transcatheter, remains the definitive treatment. Aortic regurgitation, another form of aortic valve disease, involves backward blood flow into the left ventricle and is classified as acute or chronic. Mitral valve disease encompasses mitral stenosis, primarily caused by rheumatic fever, and mitral regurgitation, the most prevalent VHD in the United States. Tricuspid regurgitation is increasingly recognized as a significant contributor to morbidity and mortality. Diagnostic tools such as echocardiography, electrocardiography, and cardiac catheterization are crucial for assessing disease severity and guiding treatment decisions. Contemporary management of VHD includes surgical interventions, transcatheter procedures, and medical therapy tailored to the specific valve condition and patient characteristics. Post procedure nursing care focuses on access site management, cardiac rhythm monitoring, and early detection of

complications. Patient education is vital for optimal outcomes, addressing the procedure, anticipated recovery, and post discharge care. As the landscape of VHD treatment evolves, a multidisciplinary approach incorporating advanced imaging, innovative procedures, and comprehensive patient care is essential for improving patient outcomes and quality of life.
Keywords: nurses, Valvular heart disease

Introduction

Valvular heart disease (VHD) exerts a profound effect on health, quality of life, and longevity. The global increase in the aging population has led to a substantial rise in the burden of VHD. Rheumatic heart disease, the most prevalent form, is observed in developing nations, whereas functional and degenerative diseases are more prevalent in higher-income countries. In the United States, life expectancy has improved due to advancements in healthcare, with the population aged over 60 years increasing from 55.7 million to 74.6 million between 2009 and 2019. Population studies indicate that the prevalence of VHD escalates with age, rising from 0.7% among individuals aged 18–45 years to 13.3% in those over 75 years of age (Baumgartner et al., 2017; d'Arcy et al., 2011). These demographic shifts have heightened the focus on managing and treating VHD, particularly its primary manifestation, heart failure (HF).

Historically, the treatment recommendations for VHD emphasized surgical interventions aimed at repairing or replacing diseased valves. Over the past two decades, innovations in minimally invasive procedures have transformed the strategies for valve repair and replacement. Catheter-based procedures, guided by evaluations of symptom status, disease severity, comorbid conditions, and the anatomy of the cardiac and vascular systems, necessitate advanced imaging interpretations. Given the complexity of these procedures, current guidelines advocate for referring VHD patients to valve centers staffed with multidisciplinary heart teams (MDTs). These teams conduct comprehensive testing, evaluation, and patient consultations to determine individualized treatment plans aligned with patient goals (Nishimura et al., 2019).

Aortic Valve Disease: Stenosis

Aortic stenosis (AoS) involves a progressive narrowing of the aortic valve (AoV), driven by an inflammatory process initiated by endothelial damage due to mechanical stress, lipid infiltration, leaflet thickening, and subsequent calcification of the valve (Joseph et al., 2017). The predominant causes of AoS include calcific disease, rheumatic disease, and congenital or acquired bicuspid valves (Osnabrugge et al., 2013). Globally, rheumatic disease is the leading cause of stenosis, while calcification of bi leaflet or tri leaflet AoVs is more common in developed countries. AoV sclerosis, which may precede significant stenosis, can be detected using echocardiography and/or computed tomography.

The prevalence of AoS increases with age, affecting approximately 1% to 2% of individuals aged 65 years and over 12% of those older than 75 years (Goody et al., 2020; Lindman et al., 2016). The prognosis for untreated severe symptomatic AoS is poor, with about 50% of patients succumbing within two years, and a five-year survival rate of approximately 20% (Joseph et al., 2017; Osnabrugge et al., 2013). AoS is categorized into four general stages based on symptoms, valve anatomy, valve hemodynamics, and associated changes in the left ventricle (LV) and vasculature (Otto et al., 2021). Diagnostic tools, including echocardiography, electrocardiography (ECG), and detailed history and physical examinations, are critical for determining the severity, disease burden, symptoms, and the presence of HF. Accurate staging and symptom assessment guide treatment and surveillance strategies.

Clinical Presentation

The hallmark symptoms of AoS include shortness of breath, angina, and syncope. Fatigue and exertional dyspnea are the most frequently reported initial symptoms, while more advanced disease often presents with orthopnea, paroxysmal nocturnal dyspnea, and edema.

These later-stage symptoms are frequently linked to LV hypertrophy and diastolic dysfunction. Symptom severity plays a pivotal role in determining the appropriate treatment approach.

Physical Examination

Key clinical findings in AoS include an evaluation of the carotid upstroke, auscultation of a systolic murmur, and signs of HF. Palpation of the carotid artery, alongside heart sound auscultation, typically reveals a synchronized pulse and heartbeat. A delayed and diminished upstroke, termed pulsus parvus et tardus, is a specific indicator of severe AoS (SAS). A characteristic high-pitched, crescendo-decrescendo midsystolic murmur is often detected at the second intercostal space along the right sternal border, with radiation to the carotids. In advanced SAS, careful auscultation may reveal a paradoxical split S2 or the eventual absence of the A2 component.

Risk and Frailty Assessment

Frailty assessments are integral to evaluating AoS patients for intervention. The Society of Thoracic Surgeons (STS) score is utilized to estimate the risk of morbidity and mortality in patients undergoing surgical procedures. However, this score does not account for the impact of frailty on surgical risk and recovery. Current treatment guidelines incorporate frailty scores into decision-making processes (Otto et al., 2021).

Diagnostic Testing

Transthoracic echocardiography (TTE) is the gold standard for diagnosing VHD, offering insights into disease morphology and severity, chamber size and function, and pulmonary status. TTE metrics classify the severity of disease as mild, moderate, or severe, forming the basis for treatment recommendations (Otto et al., 2021).

ECG findings are not diagnostic for AoS but provide information on rhythm and LV hypertrophy. For asymptomatic SAS patients, low-level stress testing can help assess symptom burden. Exercise intolerance, abnormal blood pressure responses, and other stress test findings may indicate SAS in patients who have adjusted their lifestyles to accommodate symptom progression. Gated computed tomography (CT) has emerged as the gold standard for planning transcatheter procedures, providing detailed assessments of valve sizing, calcium distribution, disease severity, and valve morphology, as well as arterial measurements critical for surgical and minimally invasive valve replacement strategies.

TREATMENT

Ongoing research aims to uncover the underlying mechanisms of progressive fibrocalcific remodelling in calcific aortic stenosis (AoS); however, no pharmacological therapy has yet been identified to prevent this progression (Goody et al., 2020). Aortic valve replacement (AVR) remains the definitive treatment for symptomatic AoS, with randomized controlled trials demonstrating its superiority over medical management. Both surgical AVR (SAVR) and transcatheter aortic valve replacement (TAVR), a less invasive catheter-based approach, are recommended for treatment. Studies have shown that TAVR using bioprosthetic valves is noninferior to surgical procedures, making it the preferred method for AVR regardless of surgical risk. Nevertheless, certain populations—such as patients under 65 years of age, those with anatomical conditions unsuitable for TAVR, or those with concomitant multivalve, coronary, or aortic disease requiring intervention—are better suited for surgical AVR.

The TAVR procedure involves the use of a catheter to deliver a collapsed bioprosthetic valve to the site of the diseased valve. Most commonly, the femoral artery is utilized as an access route to the aorta. The catheter navigates through the aortic arch and ascending aorta to the diseased valve, where the new valve is deployed via self-expansion or balloon inflation. After deployment, the catheter is removed. While TAVR was initially performed under transesophageal echocardiogram (TEE) guidance and general anesthesia, advancements in device technology have enabled the use of nurse-led conscious sedation, which has been proven safe and effective (Keegan et al., 2020).

Balloon aortic valvuloplasty (BAV) serves as a temporary palliative measure for severe symptomatic AoS. It is often utilized as a bridge to definitive treatment, such as SAVR or TAVR, or as a palliative option for patients unlikely to benefit significantly from TAVR. Symptomatic relief from BAV typically lasts 6 to 12 months, but patients continue to face high mortality risks similar to those who remain untreated.

AORTIC VALVE DISEASE: REGURGITATION

Aortic regurgitation (AR), or aortic insufficiency (AI), involves the backward flow of blood from the aorta into the left ventricle (LV) during diastole. Approximately 2% of Americans aged over 75 years have moderate to severe AR. The condition is categorized into diseases affecting the aortic valve (AoV) and leaflets or those affecting the aortic root or ascending aorta. Among patients undergoing AVR for isolated AR, aortic root diseases now represent the most common cause (Braunwald's Heart Disease, 2014). In patients under 65 years of age, congenital bicuspid AoV remains the leading cause (Peters et al., 2022). Other etiologies include endocarditis, rheumatic disease, calcific degeneration, aortic dilation, arteriosclerosis, Marfan syndrome, and syphilis.

AR is classified as either acute or chronic. Acute severe AR results in LV volume overload, diminished cardiac output, and potentially cardiogenic shock, necessitating emergent intervention. Common causes include endocarditis, aortic dissection, or trauma. Chronic AR is staged as follows: Stage A involves individuals at risk of AR; Stage B represents progressive AR with mild to moderate disease; Stage C indicates asymptomatic severe AR, where stress testing may help assess symptoms; and Stage D denotes symptomatic severe AR, with a spectrum of symptoms ranging from mild to severe.

CLINICAL PRESENTATION

Acute severe aortic insufficiency (ASAI) presents with rapid symptom progression or cardiovascular collapse. Initial presentations vary by etiology, such as severe back and chest pain in aortic dissection or fever, night sweats, and weight loss in infective endocarditis. Prompt recognition of the underlying etiology is critical for timely intervention. In contrast, chronic severe AR often remains asymptomatic until LV dilation becomes significant. Symptoms, such as dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea, typically develop gradually as the disease progresses. Late-stage manifestations include nocturnal angina and signs of left-sided heart failure, including fatigue and shortness of breath. Both compensated and decompensated AR are associated with wide pulse pressure.

PHYSICAL EXAMINATION

In ASAI, heart sounds include a low-pitched, short-duration early diastolic murmur and a soft or absent first heart sound (S1), best heard at the left third or fourth intercostal space with the patient leaning forward. Chronic AR often reveals the Austin-Flint murmur, a low-pitched, mid-diastolic sound audible at the cardiac apex. Specific physical findings in AR include Corrigan's sign (a "water hammer" pulse) and Duroziez's sign (a systolic and diastolic bruit with partial compression of the femoral artery).

DIAGNOSTIC TESTING

Transthoracic echocardiography (TTE) is essential for evaluating AR severity, LV function, and valve morphology. Advanced imaging techniques, such as transesophageal echocardiography (TEE), cardiac magnetic resonance imaging (MRI), or cardiac catheterization, assess LV size and function, AR severity, and aortic root abnormalities, including dissection or congenital anomalies.

TREATMENT

Treatment for AR depends on clinical presentation, disease severity, and chronicity. Surgical AVR remains the cornerstone of treatment for severe acute and chronic AR. Emergent intervention is critical in acute settings complicated by pulmonary edema, hypotension, or low

cardiac output. Acute AR secondary to endocarditis or aortic dissection requires stabilization, including afterload reduction, before surgery. However, beta-blockers are contraindicated in most acute AR cases due to their negative impact on compensatory tachycardia.

At present, no transcatheter aortic valve replacement (TAVR) options are commercially approved for native AR, though investigational trials are underway. For bioprosthetic valve failure, valve-in-valve TAVR is an approved intervention.

Mitral Valve Disease: Stenosis

Mitral stenosis (MS) arises from an obstruction to blood flow between the left atrium (LA) and left ventricle (LV) through the mitral valve (MV), leading to elevate LA pressure and subsequent pulmonary vascular congestion. The increased pressure can result in pulmonary hypertension, hemoptysis due to ruptured pulmonary veins, right ventricular (RV) strain, and tricuspid regurgitation (TR). The predominant cause of MS is untreated streptococcal infection, which leads to rheumatic fever and valve disease (VD); this accounts for 50% to 70% of symptomatic MS cases (Lilly, 2011). Additional etiologies include calcification of the mitral leaflets, vegetations from infective endocarditis obstructing the valve orifice, and congenital forms of MS.

The incidence of MS is low in high-income nations and has been decreasing in low- and middle-income countries. The estimated prevalence in the United States is about 1 in 100,000, with 35 cases per 100,000 in developing nations. Women constitute 80% of the cases, reflecting a higher prevalence in this group.

Clinical Presentation

As MS progresses, symptoms associated with congestion and heart failure (HF) typically prompt clinical evaluation (Grines et al., 2016). Regardless of the underlying pathophysiology (stenosis or regurgitation), patients present with fatigue, reduced exercise tolerance, shortness of breath, dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea. Other reported symptoms include chest discomfort, lower extremity edema, dizziness, or light-headedness. Symptom onset is gradual and progressive. Exacerbation of symptoms can occur with conditions or activities that elevate heart rate (HR) and cardiac output, such as fever, anemia, hyperthyroidism, pregnancy, rapid arrhythmias, emotional stress, or sexual activity.

Physical Examination

A classic auscultatory finding in MS is a low-frequency decrescendo diastolic murmur resulting from turbulent blood flow across the stenotic valve during diastole. The murmur, best heard in the left lateral decubitus position, begins after the opening snap of the MV, with its duration correlating to the stenosis severity. Palpation of the chest may reveal an RV "tap" indicative of increased RV pressure.

Diagnostic Testing

The main diagnostic modality for MS is transthoracic echocardiography (TTE), which assesses the severity of stenosis and typically reveals thickened mitral leaflets with fused commissures and restricted diastolic motion (d'Arcy et al., 2011). Disease severity is classified as mild, moderate, or severe. Transesophageal echocardiography (TEE) may be performed to better visualize the valve and LA when TTE quality is suboptimal. In select cases, cardiac catheterization or exercise stress testing may be used to clarify the diagnosis when discrepancies arise between clinical presentation and TTE findings.

Treatment

The management of MS depends on its etiology. For rheumatic MS, characterized by typical mitral valve leaflet appearance on TTE and a history of rheumatic fever, percutaneous mitral balloon valvuloplasty is recommended. This procedure, performed via the femoral vein, uses a balloon catheter to dilate the valve, improving leaflet mobility. This approach serves as first-line therapy for rheumatic MS and may provide symptomatic relief for several years. Open surgical valve replacement remains the gold standard for non-rheumatic forms of MS.

Mitral Valve Disease: Mitral Regurgitation

Mitral regurgitation (MR) is the third most prevalent form of valvular heart disease (VHD) globally, affecting approximately 24.2 million individuals, and is the most common VHD in the United States. It primarily affects older adults, with an overall prevalence of 1.7%. The prevalence increases with age, from 0.5% in individuals aged 18–44 years to 9.3% in those aged 75 years or older (Cahill et al., 2021; Peters et al., 2022).

MR occurs when blood flows back from the LV into the LA during systole. It is classified based on duration as acute or chronic. Chronic MR is further subdivided by etiology: primary/degenerative, secondary/functional, or mixed. Acute MR may result from infective endocarditis causing leaflet perforation, spontaneous chordal rupture, or myocardial infarction (MI) and can lead to rapid decompensation, necessitating intensive care and mechanical circulatory support until definitive treatment is provided.

Primary or degenerative MR (dMR) arises from structural abnormalities in the valve apparatus, such as the leaflets, chordae, papillary muscles, or annulus. These abnormalities, often linked to genetic predispositions, hinder valve closure, allowing blood regurgitation through the defective area. Secondary or functional MR (fMR), accounting for 65% of MR cases, is caused by annular dilation preventing leaflet coaptation. It is associated with dilated atria or ventricles (Aluru et al., 2022).

Clinical Presentation

Regardless of the subtype, MR presents with similar complaints, including fatigue, diminished exercise tolerance, dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea. Acute MR typically involves a rapid onset of symptoms, such as those caused by abrupt chordal rupture, whereas chronic MR progresses more gradually, with symptoms emerging as cardiac output declines. Chronic severe MR may also manifest with right-sided HF symptoms, such as peripheral edema and abdominal distension .

Physical Examination

The classic murmur of MR is a holosystolic murmur best heard at the apex with the patient in the left lateral decubitus position. In chronic MR, the murmur may radiate to the axilla or, in cases of mitral valve prolapse, to the anterior or posterior chest depending on the regurgitant jet's direction. Patients may also exhibit signs of left-sided HF, such as pulmonary rales, or right-sided HF, including jugular vein distension and peripheral edema.

Diagnostic Testing

Echocardiography, particularly TTE, is the primary diagnostic tool for MR. It evaluates LV function, pulmonary artery pressure, and MR severity and etiology. TEE is particularly useful in identifying specific causes, such as degenerative or functional MR, chordal rupture, or valvular vegetations. Although not a primary diagnostic method, cardiac catheterization may provide hemodynamic data supporting an MR diagnosis, with ventriculography quantifying regurgitation severity.

Treatment

Acute mitral regurgitation (MR), caused by chordal or papillary muscle rupture or infective endocarditis, typically necessitates surgical intervention. Stabilization with vasodilator therapy may be required preoperatively to lower systemic arterial resistance and enhance forward flow. This approach may alleviate symptoms of pulmonary vascular congestion by reducing regurgitant volume (3). For patients with acute MR deemed at prohibitive surgical risk, transcatheter therapies, such as mitral valve edge-to-edge repair (MV TEER), can serve as either a lifesaving standalone procedure or a bridge to surgery (Haberman et al., 2022).

For chronic MR, treatment strategies differ based on the underlying etiology. Functional MR (fMR) demands a distinct approach compared to degenerative MR (dMR). The primary treatment for fMR associated with left ventricular heart failure with reduced ejection fraction (HFrEF) involves optimizing guideline-directed medical therapy (GDMT). The objective of

GDMT is to restore left ventricular (LV) systolic function, facilitating normalization of the annulus size and shape to reduce MR severity. GDMT focuses on reducing afterload and managing excess fluid volume. Angiotensin-converting enzyme inhibitors (ACE-Is), angiotensin receptor blockers (ARBs), and beta-blockers are the first-line therapies for afterload reduction. Loop diuretics and mineralocorticoid receptor antagonists (MRAs) may be included to manage fluid overload.

Conversely, vasodilator therapy has no demonstrated benefit in dMR. However, GDMT is indicated in cases of systolic dysfunction or hypertension. In chronic MR, open surgical repair remains the preferred treatment for patients eligible for surgery. Advances in transcatheter therapies have provided options for patients at high or prohibitive surgical risk. MV TEER, a commercially available procedure for severe MR, is performed in a cardiac catheterization laboratory with transesophageal echocardiography (TEE) guidance under general anesthesia. The procedure employs clips to secure the anterior and posterior mitral valve leaflets, creating a double or triple orifice valve and reducing regurgitation. Up to three clips may be used during MV TEER, which has been shown to significantly reduce MR severity and improve patient quality of life.

Tricuspid Valve

The tricuspid valve has gained prominence as a focus of cardiology research and innovation. Tricuspid regurgitation (TR) is prevalent in the general population, with severe cases linked to significant morbidity and mortality (Condello et al., 2021; Fender et al., 2018). The increased incidence of TR is partly attributed to the rising use of pacemakers. Although frequently identified on transthoracic echocardiograms (TTEs), TR was historically considered benign unless associated with pulmonary hypertension, right ventricular (RV) failure, or left ventricular (LV) failure.

TR is characterized by backflow into the right atrium (RA), with varying volumes influenced by preload and structural changes. Severe TR can result in backflow into the superior and inferior vena cava. TR is classified into three types: primary, secondary, and isolated. Primary TR accounts for approximately 8% to 10% of cases and involves structural abnormalities, either congenital or acquired. Congenital causes include Ebstein's anomaly, where leaflet origins are displaced apically, often lacking chordae. Acquired causes include iatrogenic injury (e.g., biopsy-related damage, pacemaker leads), leaflet damage (e.g., endocarditis, rheumatic disease), and leaflet displacement (e.g., pacemaker leads, tumors, or chordal rupture) (Prihadi et al., 2019). Pacemaker wire-induced TR is the most common acquired cause.

Secondary TR, the more common form, results primarily from RV dilation and dysfunction, causing leaflet tethering, annular dilation, and malcoaptation. It is often associated with left-sided valvular and myocardial diseases, with severe MR affecting up to 50% and severe aortic stenosis (SAS) up to 25% of cases. Isolated TR, morphologically distinct from primary and secondary types, frequently occurs in elderly patients with atrial fibrillation. This condition leads to RA dilation, annular dilation, and leaflet malcoaptation.

Clinical Presentation

Severe TR typically manifests as symptoms of right-sided heart failure (HF) or low cardiac output. Symptoms, including fatigue, weakness, dyspnea, and exercise intolerance, may progress slowly over years.

Physical Examination

On inspection, severe TR may present with prominent, distended, or pulsatile jugular veins due to regurgitant flow into the superior vena cava. Chronic regurgitation into the inferior vena cava often leads to peripheral edema and ascites. Palpation may reveal a dynamic RV heave due to RV dilation. The liver may be enlarged, tender, and pulsatile, correlating with the TR murmur. Advanced disease may result in a taut abdomen from fluid accumulation. A

holosystolic murmur, characteristic of TR, is typically heard along the right lower and mid-sternal border with minimal radiation.

Diagnostic Testing

TTE and TEE are primary tools for assessing regurgitation severity, leaflet motion, annular dimensions, interventricular septal motion, and RA/RV size, as well as superior and inferior vena cava reflux. These findings inform prognosis and treatment strategies. Electrocardiograms (ECGs) may not show changes directly linked to TR, although abnormalities related to the underlying cause, such as RV hypertrophy in pulmonary hypertension, may be present. Right heart catheterization can assess pulmonary pressures and vascular resistance when echocardiographic data is inconclusive. Cardiac MRI can help define RV anatomy and function.

Treatment

The cornerstone of TR management is medical therapy targeting heart failure. Diuretics are the first-line treatment for fluid and volume management. For secondary TR, addressing the underlying cause—such as GDMT for LV-HF or vasodilators for pulmonary hypertension—is critical. Neurohormonal antagonists may enhance diuretic therapy by mitigating compensatory mechanisms.

Surgical repair is the gold standard for severe symptomatic TR, particularly when left-sided valve surgery is also indicated. Recent guidelines suggest individualized decision-making based on TR type and severity, RV/LV function, and underlying etiology. Isolated tricuspid surgery is recommended for symptomatic severe primary TR and asymptomatic or mildly symptomatic TR with evidence of RV dysfunction or dilation. Early surgical intervention is advised for patients with severe TR following prior left-sided valve surgery, especially if symptomatic, or asymptomatic patients showing progressive RV dilation or dysfunction.

Emerging transcatheter therapies are under investigation. While Europe has approved some transcatheter edge-to-edge repair systems, commercial approval in the United States is pending. Challenges in developing transcatheter techniques arise from the tricuspid valve's complex anatomy and surrounding structures. Current approaches include:

- **Repair**: Devices to enhance leaflet coaptation through edge-to-edge suturing or clips, or annuloplasty systems for annular size reduction.
- **Replacement**: Orthotopic valves implanted in the tricuspid position via venous access or heterotopic valves placed in caval-atrial positions to mitigate vena caval backflow without directly addressing TR.

Post procedure Nursing Care

Nursing management after transcatheter valve interventions commences in the cardiac catheterization laboratory and continues until hospital discharge. Immediate care focuses on recovery from sedation or anesthesia, ensuring hemostasis through access site management, maintaining hemodynamic stability, monitoring cardiac rhythm and perfusion, assessing neurologic status, and facilitating mobilization. Variability in care depends on the treated valve, the type of procedure, and the patient’s individual needs, all of which influence recovery outcomes.

Access Site Management

Effective access site management is a critical component of post procedure care. Commonly used access points include the femoral arteries and veins, which may be unilateral, bilateral, or both. In cases of peripheral vascular disease, alternative access points such as carotid, subclavian, axillary, or caval veins may be utilized. Catheter sizes range from 6 French to 24 French, where the French size represents the outer catheter diameter in millimeters (e.g., a 14 French catheter has a diameter of approximately 4.7 mm). Smaller French sizes result in smaller vascular punctures. Vascular closure devices are often employed with larger catheters to manage the access site. Key factors in managing the access site include catheter size,

deployment site, arterial or venous placement, and closure device usage. Prompt identification of any hemostasis or hemodynamic instability issues, along with knowledge of access characteristics, is essential for effective intervention.

Cardiac Rhythm Monitoring

The close anatomical relationship between the cardiac conduction system and heart valves poses a risk of conduction disturbances following valve procedures. These defects are most associated with aortic valve interventions and rarely observed with mitral valve procedures. For transcatheter tricuspid valve procedures, which remain in research phases, incidence data is not yet well-established. It is recommended that all patients undergoing transcatheter aortic valve replacement (TAVR) receive telemetry monitoring for a minimum of 24 hours to detect high-grade atrioventricular (AV) block. Patients with preexisting right bundle branch block or second-degree Mobitz type I AV block are at higher risk. For new or progressive conduction disturbances postprocedure, electrophysiological studies should be considered, and monitoring may need to extend to at least 48 hours. Patients experiencing new but non-progressive conduction delays during hospitalization may be discharged with mobile cardiac telemetry to detect late-onset conduction issues over a minimum of 14 days.

Pericardial Effusion

Pericardial effusion is a potential risk associated with any transcatheter valve intervention. Catheter or wire perforation may lead to bleeding into the pericardial sac, compressing the heart and restricting its function. Vigilant clinical observation is vital for early detection and management of this life-threatening complication. Signs of pericardial effusion include declining systolic blood pressure and a compensatory increase in heart rate as the left ventricle becomes compressed, reducing stroke volume. Symptoms may include chest pain, shortness of breath, or pain radiating to the back, neck, or upper abdomen. These signs necessitate prompt evaluation with transthoracic echocardiography (TTE) and escalation of care.

Medical Therapy—Heart Failure

Heart failure (HF) is a common presentation in patients with valvular heart disease (VHD). Although transcatheter procedures address the underlying valve pathology, preexisting HF persists and requires continued treatment. Adjustments to HF therapy may be necessary based on changes following the valve intervention. Optimal guideline-directed medical therapy (GDMT) is advised according to the patient's ejection fraction status, whether preserved or reduced.

For HF with reduced ejection fraction (HFrEF), GDMT includes sodium-glucose cotransporter-2 inhibitors (SGLT2is), angiotensin receptor-neprilysin inhibitors (ARNis), or angiotensin-converting enzyme inhibitors (ACE-Is) and angiotensin receptor blockers (ARBs) when ARNis are unsuitable. Additional components include mineralocorticoid receptor antagonists (MRAs) and HF-specific beta-blockers. Loop diuretics are the primary choice for fluid management, with thiazide diuretics added if response to loop diuretics is inadequate.

In heart failure with preserved ejection fraction (HFpEF; left ventricular ejection fraction ≥50%), medical therapy focuses on afterload reduction and fluid volume control. Current guidelines include SGLT2is, ARNis, ACE-Is, ARBs, and MRAs, along with management of hypertension and atrial fibrillation. The use of ARBs is recommended, while nitrates and phosphodiesterase-5 inhibitors are avoided. Loop diuretics remain central to fluid management, with thiazide diuretics added for refractory cases.

Medical Therapy—Antiplatelet and Anticoagulation Therapy

The use of antiplatelet therapy after transcatheter procedures was initially guided by protocols from early research studies. Early transcatheter aortic valve replacement (TAVR) protocols incorporated dual antiplatelet therapy (DAPT) with clopidogrel and aspirin to mitigate the risk of thrombus formation on the valve leaflets or devices, which could increase

the likelihood of stroke. Current guidelines, as outlined in the TAVR expert consensus decision pathway, still recommend DAPT. However, recent meta-analytic evidence suggests that single antiplatelet therapy following TAVR is associated with a reduced risk of bleeding without an increased incidence of ischemic events.

Patient Education

Patient education, whether for surgical or minimally invasive procedures, is a vital component of care and includes both pre- and postprocedure guidance for patients, families, and caregivers. Effective education equips patients and their support systems with knowledge about the procedure, anticipated hospital course, and postdischarge care, fostering optimal outcomes. It also helps identify resources to overcome potential barriers and addresses both tangible and perceived challenges.

Conclusion

Nurses play an indispensable role in the comprehensive management of valvular heart disease, particularly following transcatheter and surgical interventions. Their expertise spans from immediate postprocedural care, including hemostasis and hemodynamic monitoring, to addressing complications like conduction disturbances and pericardial effusion. Moreover, their involvement extends to patient education, a cornerstone for successful recovery and long-term management. By integrating advanced therapeutic protocols, tailoring care to patient-specific needs, and promoting education, nurses not only enhance clinical outcomes but also contribute to improving patients' quality of life. As cardiology continues to evolve, the pivotal contributions of nurses will remain integral to advancing care standards and fostering recovery in this complex patient population.

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