12 Screening and Diagnosing Cardiovascular Disease in Chronic Kidney Disease

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Before You Start: Facts You Need to Know

- Cardiovascular disease is a leading cause of morbidity and mortality in patients with chronic kidney disease as determined by reduced estimated glomerular filtration rate and/or albuminuria.
- Patients with chronic kidney disease are known to have increased risk of coronary artery disease, heart failure, arrhythmia, valvulopathies, and sudden cardiac death.
- Atherosclerosis is both accelerated in development and in calcification in patients with chronic kidney disease.
- Heart failure is the most common symptomatic manifestation of cardiovascular disease requiring hospitalization in patients with chronic kidney disease.
- Blood B-type natriuretic peptide, N-terminal pro B-type natriuretic peptide, galectin-3, and soluble ST-2 are approved tests as these aid in the diagnosis, prognosis, and management of heart failure; however, caution should be exercised in the interpretation of these markers in the setting of chronic kidney disease.
- Aortic valve sclerosis and mitral annular calcification are common valve pathologies associated with chronic kidney disease.
- All forms of arrhythmias are more common in chronic kidney disease, especially sudden death which is markedly increased in risk in dialysis patients.

12.1 Why Screening for Cardiovascular Disease Is Important in Chronic Kidney Disease

Screening is a strategy which help us to identify people who have risk factors (primary prevention) or occult pathologies (secondary prevention) so that early intervention and treatment can be offered, the natural history of a disease process can be altered, and disease outcomes can be improved. Cardiovascular disease (CVD) is leading cause of morbidity and mortality in the world and accounts one third of all deaths. CVD is also a leading cause of morbidity and mortality in chronic kidney disease (CKD) patients. Patients with CKD are known to have increased risk of coronary artery disease (CAD), heart failure, arrhythmia, valvulopathies, and sudden cardiac death [1]. According to The United States Renal Data System (USRDS) 2022 annual report, CVD of any type was present in 75.8% of patients receiving hemodialysis, 65.4% of patients

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receiving peritoneal dialysis, and 52% of patients with a kidney transplant. Again, according to the same report, CVD was found to be responsible for more than half of the deaths in both hemodialysis and peritoneal dialysis patients [2]. In end stage renal disease (ESRD) population, mortality due to CVD is 20–30 times higher than general population. This increased risk is not limited to ESRD population, but it is seen in all stages of CKD. In a population based study including 1,120,295 adults, it is shown that cardiovascular events increased inversely with estimated glomerular filtration rate (eGFR) [3]. In a meta-analysis reviewing 39 studies involving 1,371,990 non-dialysis dependent CKD patients, it was shown that non-dialysis dependent CKD was associated with increased risk of cardiovascular death [4]. In the light of above information, screening and early diagnosing of CVD is very important in CKD population.

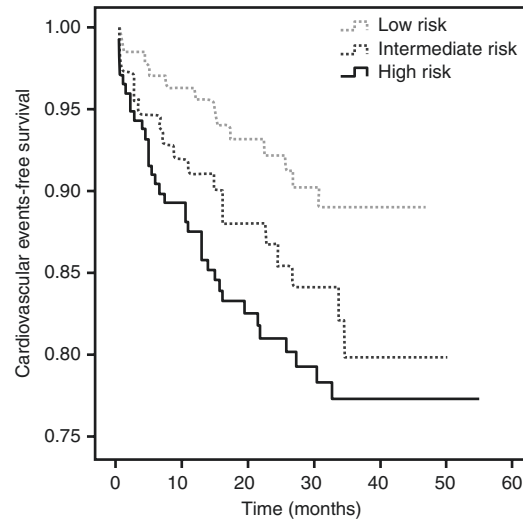


Fig. 12.1 Event-free survival from major cardiac events according to the Framingham risk score applied to a population of patients with chronic kidney disease

12.2 What Are the Approaches to Screen for Coronary Artery Disease?

Chronic kidney disease itself is an independent risk factor for the development of CAD and CAD is the leading cause of morbidity and mortality in this patient group. All adult patients including those with CKD should undergo an assessment for CAD risk using a standard risk assessment such as that proposed by the Framingham investigators [5]. Variables in the Framingham risk calculation include age, total or low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, smoking, and systolic blood pressure [6]. A 20% 10-year risk (2% annual risk) of nonfatal myocardial infarction or cardiovascular death is considered high risk and is a call for full prevention measures in the general population. Most patients with CKD (67%) will be in Framingham moderate- or high-risk groups; however, as shown in Fig. 12.1, patients with Stages 3–5 CKD in these groups will have a 10–20% annual risk of cardiovascular events (tenfold that of subjects in Framingham) [5]. Therefore; traditional prognostic tools such as the Framingham score have limited prognostic power as traditional risk factors fail to fully explain the increased risk in CKD patients [7]. Also, classical signs and symptoms of CAD may not be observed in CKD and especially in ESRD patients and it is more difficult to correctly diagnose the acute coronary syndrome in these patient groups than normal population. There are several reasons that can explain this situation such as lower sensitivity to chest pain (angina), specific electrocardiogram (ECG) changes are seen in a relatively small proportion of patients with angina, CAD symptoms may be incorrectly attributed to other CKD complications and serum biomarkers related to CAD might be chronically elevated in the absence of acute coronary syndrome (ACS). Serum biomarkers especially troponin assays (both high-sensitivity troponin I and troponin T) may be used for risk stratification and may be helpful for detecting asymptomatic CAD. Although elevated values are less definitive, dynamic change in troponin levels may be useful for myocardial infarction (MI) diagnosis and a normal troponin assay may be sufficient to rule out infarction. But we need more data to interpret troponin levels for management decisions [8].

We can use exercise stress testing for exercise prescription and prognosis in high-risk individu-

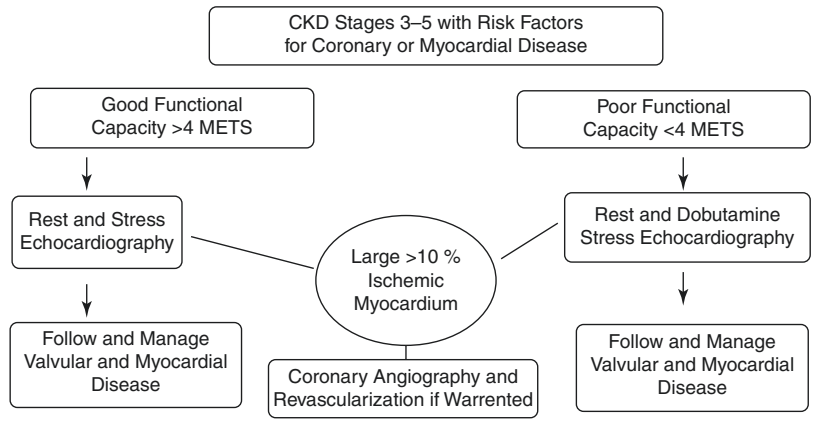


Fig. 12.2 Coronary artery disease screening algorithm (METS metabolic equivalents of functional capacity)

als. Exercise ECG has limited role due to high rates of abnormal baseline ECG, left ventricular hypertrophy, and conduction abnormalities. The other factor limiting the role of exercise ECG is the reduced exercise capacity commonly seen in CKD and especially ESRD patients [9]. Therefore, exercise stress testing combined with either echocardiographic imaging or nuclear scintigraphy is reasonable.

For those who cannot exercise, both dobutamine and dipyridamole/adenosine/regadenoson can be used as pharmacological means of achieving myocardial perfusion imaging. Large areas of ischemia (>10% of the left ventricular myocardium) usually call for invasive assessment of coronary lesions and consideration for revascularization. In the setting of diabetes and multivessel disease, coronary artery bypass surgery is the preferred method of revascularization [10]. Coronary computed tomography angiography in patients with CKD is not advised given the very high rates of coronary calcification which causes "bloom" artifact which works to make lesion severity difficult to assess [11]. However, if vascular calcification is detected incidentally on computed tomography or roentgenography, it is indicative of advanced atherosclerosis, and attention should be paid to both atherosclerosis risk factors and the elements of CKD mineral and bone disorder (phosphate retention, hyperparathyroidism, and relative hypocalcemia) (Fig. 12.2) [12, 13].

12.3 Should Patients with Chronic Kidney Disease Undergo Routine Echocardiography?

According to 2022 cardiology guidelines; heart failure (HF) is defined as a complex clinical syndrome with symptoms and signs due to any structural and/or functional disorder [14]. In CKD patients, it is difficult to distinguish classic HF symptoms and signs such as fatigue, edema, effort intolerance from symptoms related to volume overload [15]. We also know the very high incidence of left ventricular hypertrophy, risk for Stage A and Stage B heart failure in CKD patients and the associations between CKD and valvular heart disease. Therefore; all patients with CKD should be considered for echocardiography at the time CKD is diagnosed by the presence of reduced estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m^2 or evidence of kidney damage manifest by an increased urine albumin: creatinine ratio or imaging evidence of kidney disease such as polycystic kidneys by ultrasound [16]. Importantly, cardiovascular disease including coronary disease and heart failure occurs at much earlier ages than in the general population [17]. The presence of combined heart and kidney failure is considered as "cardiorenal syndrome" and should be considered in the context of the more antecedent abnormality with respect to both diagnosis and management [18]. Five subtypes of cardiorenal syndromes are dis-

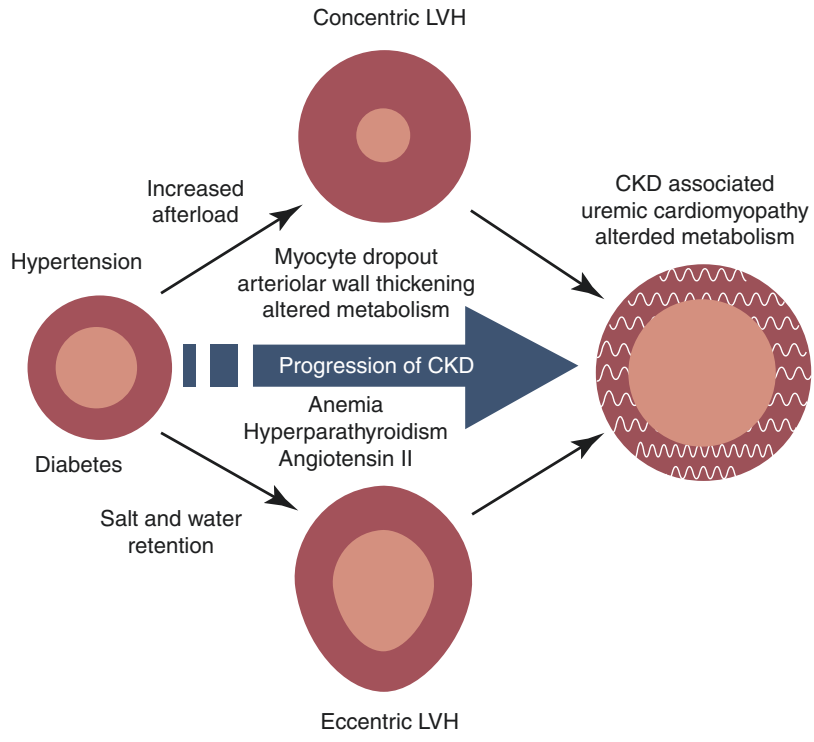


Fig. 12.3 Hypertension, diabetes mellitus, and the development of chronic kidney disease cardiomyopathy

played in (Box 12.1). The current Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines recommend echocardiograms for all CKD 5D patients 1–3 months after renal replacement therapy initiation and at 3-year intervals thereafter [19]. Serial echocardiographic examination at closer intervals such as 12 months may provide additional benefits in terms of prognosis. (Boxes 12.2 and 12.3). Echocardiography with complete Doppler assessment reliably estimated left ventricular ejection fraction (normal 55–75%), left ventricular hypertrophy (left ventricular mass index >115 and >95 g/m^2), and assesses both the morphology and flow characteristics of all four cardiac valves. According to recent studies; left ventricular hypertrophy and diastolic dysfunction are the most common structural and functional defects in hemodialysis patients, respectively [20]. Findings suggesting reduced ejection fraction, diastolic dysfunction, or regional wall motion abnormalities may prompt an evaluation for chronic cardiac ischemia as discussed above [21]. Echocardiographic evaluation of left ventricular diastolic dysfunction (LVDD) can be complicated. Especially six parameters are basically used for diagnosis and grading of LVDD. These are E wave, E/A ratio, septal or lateral è, average E/è, left atrial volume index, and peak tricuspid regurgitation velocity [22]. E and A represent velocities of the rapid early and late transmitral diastolic flow, while è is a measurement of mitral annulus recoil velocity. Diastolic dysfunction is ideally graded according to the European Association of Cardiovascular Imaging/American Society of Echocardiography criteria as normal, Grade I (impaired relaxation and decreased suction of the LV), Grade II (pseudonormalization, increased stiffness of the LV, and possible elevated filling pressure), and Grade III (most severe form) with restrictive filling with elevated filling pressure and noncompliant LV [23]. Chronic kidney disease is associated with a form of uremic or CKD cardiomyopathy as shown in Fig. 12.3. The cardiomyopathy associated with CKD is characterized by the presence of left ventricular hypertrophy, evidence of diastolic dysfunction, and, in more severe cases, superimposed systolic dysfunction with reduced

ejection fraction. The structural remodeling of the heart due to diffuse interstitial fibrosis and cardiac hypertrophy can cause electromechanical dysfunction and an increased risk of sudden cardiac death [24]. Cardiac magnetic resonance imaging (MRI) is the gold standard for LV mass quantification, chamber size and volume. But the use of contrast enhanced MRI in advanced CKD patients is limited due to the potential increased risk of gadolinium retention and nephrogenic systemic fibrosis. This risk can be decreased with the use of macrocyclic MRI contrast agents or using non-contrast tissue characterization techniques [25].

Box 12.1 Five Cardiorenal Syndromes and Their Common Clinical Scenarios

Cardiorenal Syndrome (CRS) *General Definition*

A pathophysiological disorder of the heart and kidneys whereby acute or chronic dysfunction in one organ may induce acute or chronic dysfunction in the other organ.

CRS Type I (Acute *Cardiorenal Syndrome*).

Abrupt worsening of cardiac function (e.g., acutely decompensated congestive heart failure) leading to acute kidney injury.

CRS Type II (Chronic *Cardiorenal Syndrome*).

Chronic abnormalities in cardiac function (e.g., chronic congestive heart failure) causing progressive and permanent chronic kidney disease.

CRS Type III (Acute Renocardiac *Syndrome*).

Abrupt worsening of kidney function (e.g., acute kidney injury) causing acute cardiac disorder (acute heart failure).

CRS Type IV (Chronic Renocardiac *Syndrome*).

Chronic kidney disease (e.g., diabetic nephropathy) contributing to decreased cardiac function and cardiac hypertrophy and fibrosis and/or increased risk of adverse cardiovascular events.

CRS Type V (Secondary *Cardiorenal Syndrome*).

Systemic conditions (e.g., sepsis) causing both acute cardiac and renal injury and dysfunction.

Box 12.2 What the Guidelines Say You Should Do

- Patients with chest pain should receive a complete history and physical examination to assess the probability of coronary disease before additional testing.
- A resting ECG is recommended in patients without an obvious, noncardiac cause of chest pain.
- Assessment of resting left ventricular function and evaluation for abnormalities of myocardium, heart valves, or pericardium are recommended with the use of Doppler echocardiography in patients with known or suspected coronary disease and a prior MI, pathological Q waves, symptoms, or signs suggestive of heart failure, complex ventricular arrhythmias, or an undiagnosed heart murmur.
- Standard exercise stress testing is recommended for risk assessment in patients with stable coronary disease who have an interpretable ECG and no disabling comorbidity. Pharmacological stress with nuclear myocardial perfusion imaging or echocardiography is an alternative in those who are incapable of exercising to an accepted workload.
- Echocardiograms should be performed in all patients at the initiation of dialysis, once patients have achieved dry weight (ideally within 1–3 months of dialysis initiation), and at 3-yearly intervals thereafter.
- In asymptomatic patients with stable coronary artery disease and chronic kidney disease, routine angiography and revascularization are not recommended.

- An initial invasive strategy did not demonstrate a reduced risk of clinical outcomes or improved quality of life measures compared with an initial conservative strategy in stable patients with moderate CKD and at least moderate ischemia.
- Coronary computed tomography angiography is reasonable for patients with a low to intermediate pretest probability of ischemic heart disease who have a disabling comorbidity.

Source: Data from Refs. [1, 26–28].

Box 12.3 Relevant Guidelines

1. American Heart Association Guidelines:
   (a) 2012 ACCF/AHA/ACP/AATS/PCNA/SCAI/STS guideline for the diagnosis and management of patients with stable ischemic heart disease: executive summary. A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, and the American College of Physicians, American Association for Thoracic Surgery, Preventive Cardiovascular Nurses Association, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. Circulation. 2012; 126:3097–137 [26].
   (b) 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines J Am Coll Cardiol. 2021 Nov, 78 (22) e187–e285 [27].
   (c) 2021 ACC/AHA/SCAI Guideline for Coronary Artery Revascularization: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines Circulation. 2022;145: e18–e114 [28].
2. National Kidney Foundation Guidelines:
   (a) National Kidney Foundation. K/DOQI clinical practice guidelines for cardiovascular disease in dialysis patients. Am J Kidney Dis. 2005;45 Suppl 3:S1–154 [1] [18].

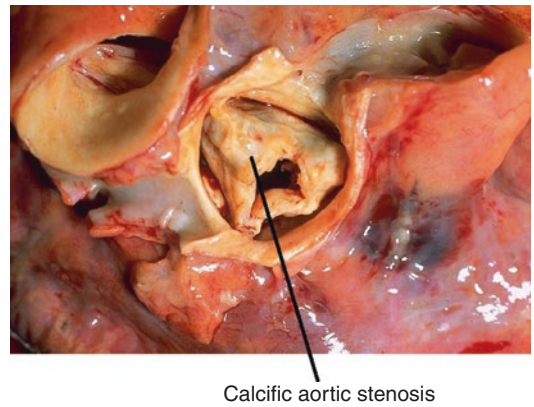


Fig. 12.4 Calcific aortic stenosis

A finding of significant valvular or pericardial disease warrants clinical correlation and follow-up. Most patients with moderate or more aortic stenosis/regurgitation or mitral regurgitation will require annual echocardiography and cardiology consultation for surveillance. In general, severe symptomatic aortic stenosis (Fig. 12.4) and/or regurgitation is an indication for valve replacement [12].

Pericardial disease may develop in kidney failure as pericarditis, pericardial effusion, or chronic constrictive pericarditis. BUN elevations over 60 mg/dL may lead to inflammation in the pericardial membranes causing uremic pericarditis. Fluid overload can also lead to pericardial

inflammation without uremia. Typical symptoms include fever and pleuritic chest pain that is relieved by sitting up or bending forward. Platelet function impairment may cause a hemorrhagic pericardial effusion and possibly tamponade depending on the rate of fluid accumulation. Typical diffuse ST elevations observed with acute pericarditis are generally not shown when uremia is the cause [29]. Echocardiography can exclude silent effusions and useful in determining associated myocarditis and altered ventricular function. Early echocardiography at the time of initiation of dialysis can also be beneficial for pericardial disease.

12.4 What Blood Biomarkers Are Useful in Heart Failure?

The role of biomarkers has consistently increased in the current medical practice due to their contribution to diagnosis, prognosis, and treatment. An ideal biomarker should be easily available and interpretable, cheap, rapid, accurate and specific for a particular situation [30]. There are many potential biomarkers for heart failure. The natriuretic peptides are the most extensively studied and used biomarkers for heart failure.

Both blood B-type natriuretic peptide (BNP) and N-terminal pro B-type natriuretic peptide (NT-proBNP) have been approved, recommended by guidelines, and are commercially available for several years. When measured in blood, they are indicated as diagnostic aids for the evaluation of patients with acute shortness of breath, prognostic indicators for death and heart failure hospitalization, and aids in the management of patients particularly with respect to the titration of chronic medications. In general, when BNP >200 pg/mL and NT-proBNP>2000 pg/mL, there is increased myocardial production even in the presence of reduced clearance by the kidneys. The higher the levels, the greater the positive predictive value for heart failure and the worse the prognosis for hospitalization or death. Chronic use of angiotensin-converting enzyme inhibitors, angiotensin receptor antagonists, aldosterone receptor blockers, and beta-adrenergic receptor antagonists and use of biventricular pacing have been shown to reduce BNP/NT-proBNP over time. In approximately 25% of patients with preserved kidney function, natriuretic peptides can be normalized (BNP <100 pg/mL, NT-proBNP<150 pg/mL) with therapy for heart failure. In the setting of CKD, it is rare for natriuretic peptides to normalize; however, relatively lower levels (~50% reduction from prior levels) are associated with a favorable prognosis. Conversely, a doubling of levels over a time frame of 6 weeks or more portends a high rate of future hospitalization and death, both from pump failure and arrhythmias.

Mid-regional proatrial natriuretic peptide (MR-proANP) is a new marker and it can be useful for diagnosis and prognosis of heart failure in CKD patients. Cut-off values for the diagnosis of heart failure increased with the decreased glomerular filtration rate. However, there is no large-scale study in CKD patients to identify the threshold more precisely [31].

Galectin-3 is a paracrine substance produced by macrophages that are participating in myocardial fibrosis. Increased levels of galectin-3 (>25.9 ng/mL) are strongly prognostic for short-term death and hospitalization in patients with either diastolic or systolic dysfunction. There have been very limited number of studies evaluating the clinical value of galectin-3 in patients with CKD; however, many subjects in the heart failure studies where it was measured met the criteria for CKD according to eGFR <60 mL/min [32]. A recent study which included asymptomatic hemodialysis patients showed that galectin-3 was associated with cardiovascular mortality [33]. Another study which also includes hemodialysis patients also showed the association between galectin-3 and cardiac mortality [34]. A suggested algorithm for the management of heart failure using galectin-3 is shown in Fig. 12.5.

Soluble ST2 (sST2) and interleukin-33 compete for the transmembrane protein ligand (ST2L) and induce production of T helper type 2 cytokines. In heart failure, serum ST2 is elevated and indicates increased abnormal immune cell signaling related to myocardial dysfunction. ST2 aids in prognostication in patients with acute and

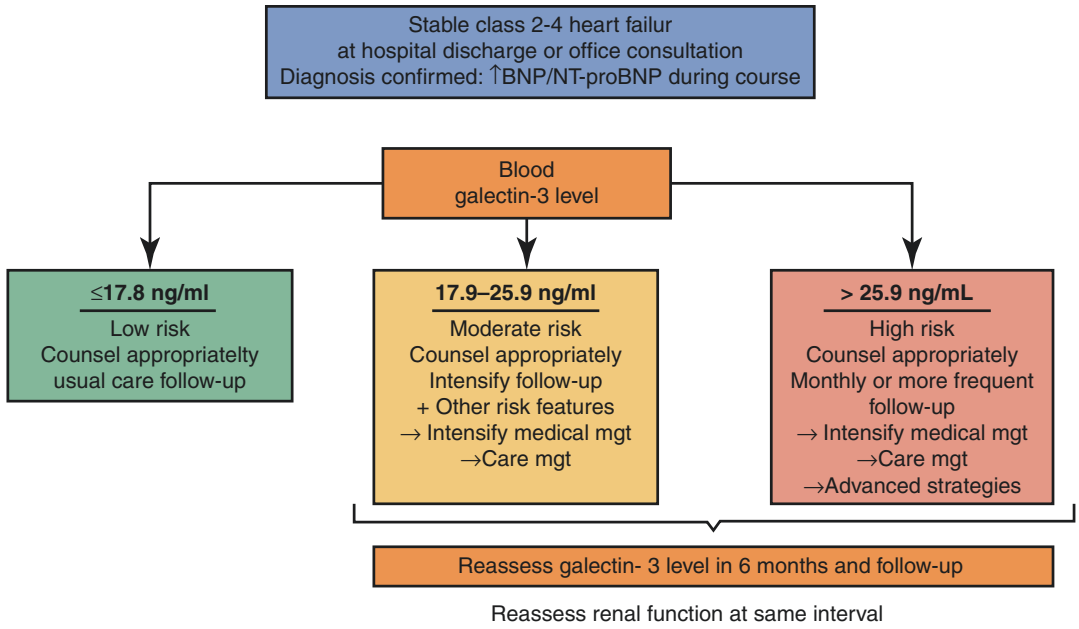


Fig. 12.5 Suggested algorithm for the management of heart failure patients using galectin-3 levels measured in blood

chronic heart failure, particularly when at very high levels (sST2 > 36.3 ng/mL). However, an elevated concentration of serum sST2 is found in CKD patients and correlates with progression of CKD [35]. Serum sST2 may be also associated with secondary hyperparathyroidism. The sST2 may have an important role in the development of CKD or as a marker of disease severity, particularly in those with incipient heart failure. Future research in this area is warranted.

Growth differentiation factor-15 (gdf-15) is a member of transforming growth factor β superfadf-15 secreted by myocardial cells due to ischemia, inflammation, and oxidative stress and it helps myocardial repair. Serum gdf-15 gradually increases with the decrease of glomerular filtration rate. Its cut-off level in CKD patients was found as 1646 ng/L. It can help the diagnosis of diastolic dysfunction and heart failure in CKD patients. It was also shown that higher serum levels of gdf-15 were associated with cardiovascular events in CKD patients [31]. Combined use with other markers may increase its prognostic role. However, more extensive studies are needed to confirm its usefulness. High-sensitivity troponin T (hs-TnT) may also be used as a predictive factor for heart failure in CKD patients. In a recent study including old patients, most of whom had renal dysfunction, patients with hs-TnT < 5 ng/L had lower heart failure risk [36]. There is also need for further studies for the use of this biomarker. Heart-type fatty acid-binding protein (H-FABP) is another promising marker with very limited number of studies in CKD patients. Higher serum level of H-FABP is associated with adverse cardiovascular events in heart failure patients [37].

12.5 Should Patients with Renal Dysfunction Have Arrhythmia Surveillance?

Maintenance of normal sinus rhythm can become progressively more difficult in patients with CKD who develop left ventricular hypertrophy, left atrial dilatation, right ventricular strain and hypertrophy, and right atrial dilatation. With activation of factors that promote cardiac fibrosis, the conduction system of the heart can show signs of failure at all levels. Thus, at the minimum in an asymptomatic patient with CKD, a 12-lead electrocardiogram should be obtained on an annual basis and with any change in cardiac symptoms.

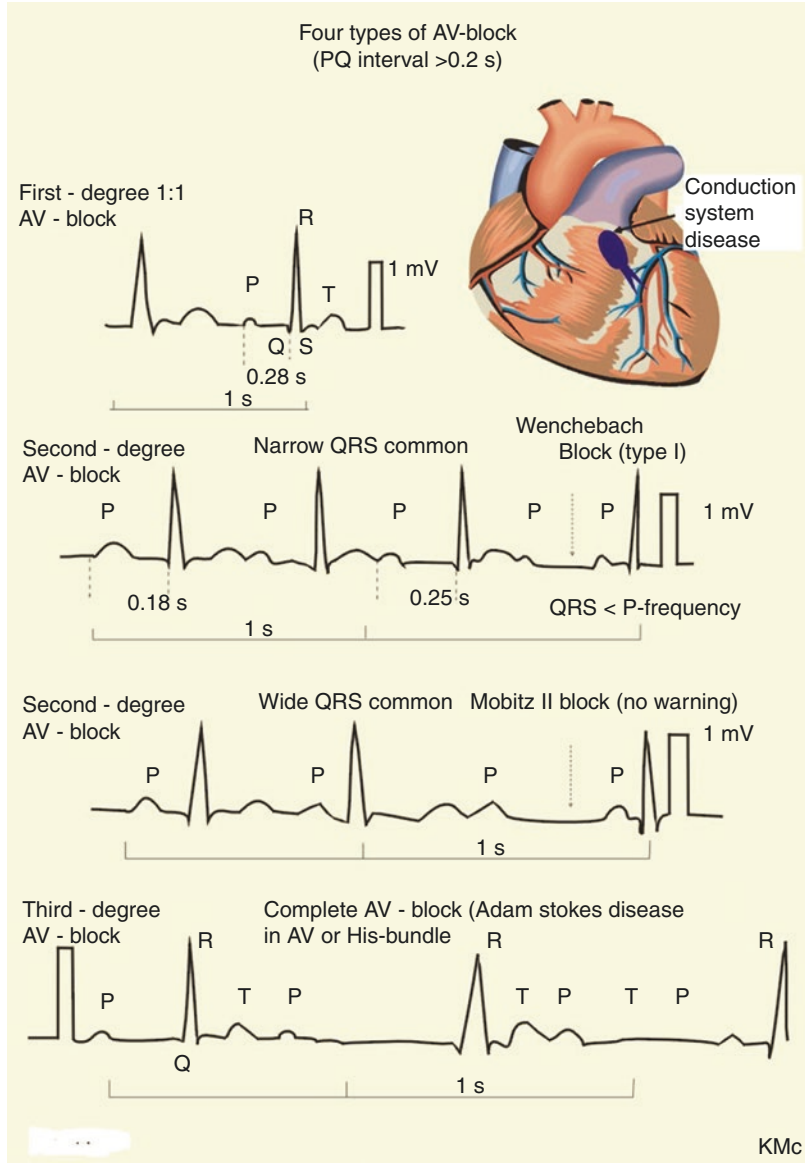
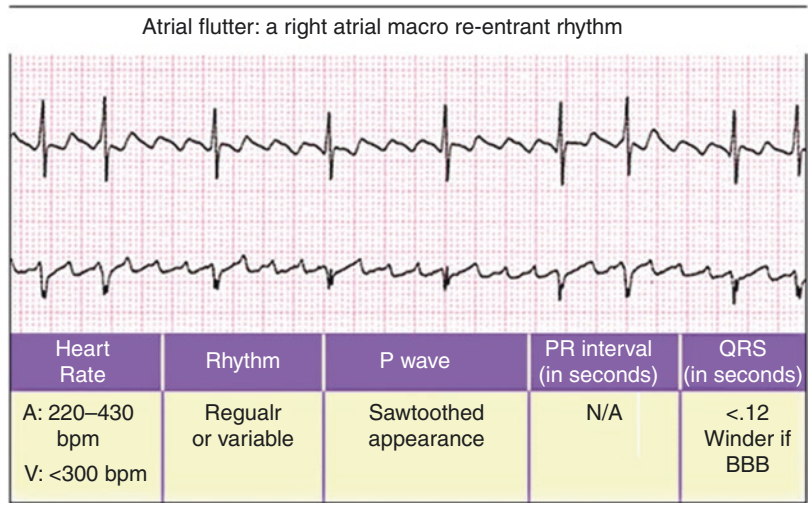


Fig. 12.6 Types of atrioventricular block as identified by electrocardiography

Failure of conduction at the level of the sinus node can lead to sick sinus syndrome (episodes of sinus pauses and tachycardia), atrioventricular node block (Mobitz Type II second degree and complete heart block (Fig. 12.6), and bundle branch blocks. These lesions in symptomatic patients are indications for permanent pacemaker implantation.

Right atrial dilatation can create a macro reentrant circuit which facilitates atrial flutter. This rhythm is recognized by sawtooth atrial depolarization waves and ventricular conduction typically in a 2:1 or 3:1 ratio (Fig. 12.7). Atrial flutter is easily managed by radio-frequency ablation and deserves electrophysiology referral. Left atrial dilatation and left ventricular hypertrophy as well as advanced age and hypertension are strong determinants for the development of atrial fibrillation (AF). Atrial fibrillation is the most common dysrhythmia among general and CKD populations. The prevalence of AF is approximately 15–20% in CKD patients not on dialysis and 15–40% in patients on dialysis [38]. Because the disorganized rhythm leads

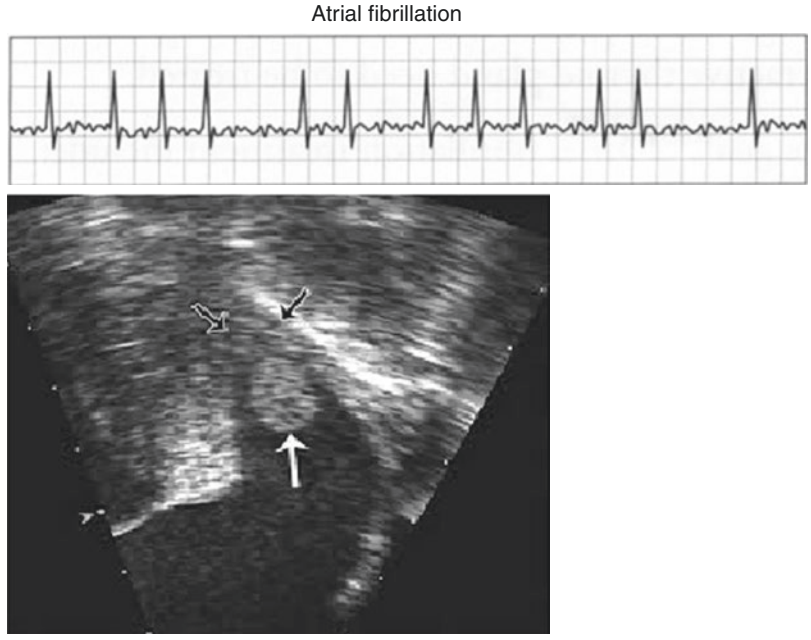
Fig. 12.7 Atrial flutter



Heart Rate	Rhythm	P wave	PR interval (in seconds)	QRS (in seconds)
A: 220–430 bpm V: <300 bpm	Regualr or variable	Sawtoothed appearance	N/A	<.12 Winder if BBB

A=atrial, V=ventricular
BBB=bundle branch block

Fig. 12.8 Atrial fibrillation on electrocardiography and a left atrial appendage identified by transesophageal echocardiography



to stasis of blood in the left atrial appendage, thrombi can form and be ejected into the left circulation resulting in stroke and systemic cardioembolism (Fig. 12.8). Thus, AF presents multiple management dilemmas including rhythm versus rate control, anticoagulation, and heart failure prevention. Any patient who presents with palpitations, tachycardia, or stroke symptoms should be assessed for AF with inpatient monitoring, 24- or 48-h outpatient Holter monitoring, or patient-triggered event monitoring. For difficult cases, an implantable loop recorder can be placed subcutaneously in the infraclavicular region and give information about cardiac rhythm for several years using noninvasive computer interrogation. In the setting of cryptogenic stroke, use of intensive rhythm monitoring has shown that approxi-

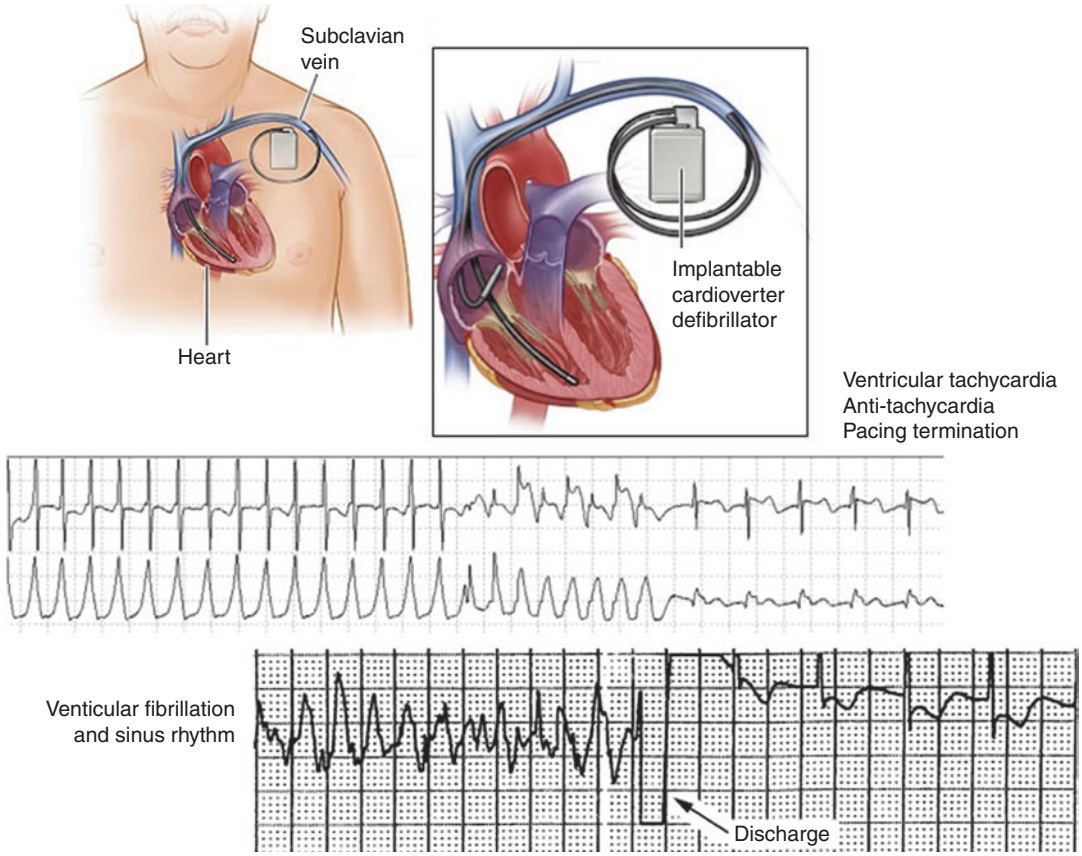


Fig. 12.9 Implantable cardio-defibrillator and demonstration of its two major forms of therapy: (1) anti-tachycardia pacing termination of ventricular tachycardia and (2) defibrillation for ventricular fibrillation

mately one third of cases can have the stroke be attributable to paroxysmal AF that was previously unrecognized.

Approximately 35–45% of CKD patients have also ventricular arrhythmias in the form of ventricular extrasystole, non-sustained and sustained ventricular tachycardia, and ventricular fibrillation. Ventricular arrhythmias may manifest as palpitation, syncope, and chest pain. If not recognized, sudden cardiac death may occur as first manifestation. ECG and 24 h ECG monitoring are important for diagnosis and risk assessment. Echocardiography and cardiac MRI can also help for detection of structural heart disease which is one of the underlying causes of arrhythmias [39].

Sudden cardiac death is typical sudden natural death, thought to be of cardiac origin, occurring within 1 h of onset of symptoms in witnessed cases, and within 24 hour of last being seen alive without witnessing [40]. Sudden cardiac death is the leading cause of death in CKD and ESRD. The details surrounding these cases are often difficult to pull together since many occur in the home and out of hospital. Presumably heart block, electromechanical dissociation, pump failure, or ventricular fibrillation is the terminal scenario. The implantable cardioverter-defibrillator has no role for primary prevention but patients with left ventricular ejection fractions <35%, those with a history of a prior resuscitated cardiac arrest, and spontaneous sustained ventricular tachycardia on monitoring should all be considered for implantable cardioverter-defibrillators. These devices reduce cardiac mortality in the general population but have not definitively been shown to prolong survival in patients with CKD or ESRD. The two major therapies delivered by implantable cardio-defibrillators are anti-tachycardia pacing and defibrillation as shown in Fig. 12.9. Because of increased myocardial

interstitial matrix in CKD and left ventricular hypertrophy, CKD and ESRD patients can be expected to have higher defibrillation thresholds and should undergo more frequent monitoring by the electrophysiologist using noninvasive programmed stimulation [41].

12.6 Summary

High rates of serious cardiovascular disease in patients with CKD and ESRD call for a more attentive approach to both routine and responsive testing in patients at risk or with potential cardiac symptoms [42]. The nephrologist needs a basic understanding of electrocardiographic interpretation both on routine single-lead monitoring and with 12-lead electrocardiography. Use of stress imaging, echocardiography, and continuous forms of rhythm monitoring provide an approach for the diagnosis and management of cardiovascular disease. Early detection and prompt management offer the hope for prevention of myocardial infarction, heart failure, valvular-induced structural damage and fatal arrhythmias.

Before You Finish: Practice Pearls for the Clinician

- Assess atherosclerosis risk factors on all patients and work to manage them to optimal levels.
- Serum biomarkers especially troponin assays may be used for risk stratification and may be helpful for detecting asymptomatic CAD. Although elevated values are less definitive, dynamic change in troponin levels may be useful for myocardial infarction (MI) diagnosis and a normal troponin assay may be sufficient to rule out infarction.
- Exercise stress testing combined with either echocardiographic imaging or nuclear scintigraphy is reasonable due to limitation of exercise stress testing in CKD patients.
- Diagnose significant cardiac ischemia with stress imaging. Large amounts of ischemia (>10% of the left ventricle) deserve coronary angiography and consideration of revascularization.
- Obtain routine 12-lead electrocardiography and have a low threshold to obtain more advanced forms of monitoring in patients with palpitations, near syncope, syncope, and stroke.
- Consider echocardiography for all patients with CKD and ESRD for assessment of myocardial function and valvular disease. Echocardiography is recommended for all CKD 5D patients 1–3 months after renal replacement therapy initiation and at 3-year intervals thereafter. We also recommended serial echocardiographic examination at closer intervals such as 12 months may increase prognostic value. All patients with considerable abnormalities need cardiology consultation and surveillance.
- In acute or chronic dyspnea, or when heart failure is suspected, elevated levels of BNP, NT-proBNP are recommended to support the diagnosis of heart failure and can portend decompensation and death. New markers such as galectin-3, ST2, MR-proANP, gdf-15 may also be used for these purposes if there is access to them.
- Patients with left ventricular ejection fractions <35%, a history of a prior resuscitated cardiac arrest, and spontaneous sustained ventricular tachycardia on monitoring should all be considered for implantable cardioverter-defibrillators.

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