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Electrocardiographic abnormalities among late-stage non-dialysis chronic kidney disease patients

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Abstract

Objectives: Cardiovascular disease (CVD) complication is common among chronic kidney disease (CKD) patients. Thus, knowledge about CVD and ECG abnormalities in CKD are essential due to progressive nature of the disease and increased risk of sudden cardiac death. This study aims to scrutinize the ECG abnormalities among nondialysis late-stage CKD patients.

Methods: A descriptive observational study was conducted at Dr. Soetomo General Hospital, Surabaya, Indonesia. Subjects were hospitalized patients with late-stage CKD between 1 January and 31 December 2019, who were consulted at the department of cardiology and vascular medicine during their initial admission at emergency room. ECG interpretation for this study was done by qualified cardiologist.

Results: There were 191 patients included in this study. Mean ages were 52.2 ± 11.8 years old and 51% were males. Total 143 (74.9%) patients had anemia, 111 (58.1%) had hypertension and 75 (39.3%) had type 2 diabetes mellitus. Mean serum creatinine was 10.5 ± 8.0 mg/dL. There were 176 (92.1%) patients with at least one form of ECG abnormalities. Prolonged QTc interval (36.6%), fragmented QRS complex (29.8%), poor R wave progression (24.6%), peaked T wave (22.0%) and left ventricular hypertrophy (16.7%) were the most common abnormalities.

Conclusions: ECG abnormalities are common among nondialysis late-stage CKD patients. Given the fact that long-term CKD influences the pathogenesis cardiovascular diseases and substantial cardiovascular mortality, there is a need to screen Indonesian CKD patients who are at risks of getting earlier complications.

Keywords: cardiovascular disease; chronic kidney diseases; electrocardiogram.

Introduction

Chronic kidney disease (CKD) is described as a kidney abnormalities stages, both structure and/or function, persist for more than 3 months, with many health implications [1]. CKD still becomes a major public health problem. Estimated global prevalence in CKD is consistent between 11 and 13% with the majority of stage 3 [2]. In Indonesia, CKD was found in 7.5% subjects with hypertension (HTN), proteinuria and/or diabetes [3].

CKD has a strong association with increased rates of hospitalization, morbidity and mortality [4]. CKD also increases the risk of cardiovascular disease (CVD) even from early stage before ESRD. Cardiovascular complication is the highest cause of mortality in late-stage CKD patients, this rate higher in dialysis patients than nondialysis [5, 6]. In patients undergoing dialytic therapy, sudden cardiac death (SCD) accounts for approximately 60% of cardiac-related deaths, and the rate of mortality due to cardiac arrest is 93 events/(1000 patients/year) in the first year of dialysis [7].

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Although modern diagnostic technology is highly developed, electrocardiogram (ECG) is still a simple, non-invasive and useful diagnostic tool for diagnosing the majority-asymptomatic progressive nature of CVD morbidity and mortality in late-stage CKD patients [8, 9]. In limited setting, ECG is able to detect heart rhythm disturbances, cardiac conduction abnormalities, chambers abnormalities and sign of myocardial ischemia [4]. Abnormalities of ECG that occur in late-stage CKD patients can be independently estimate future CVD and risk of sudden death. However, various kinds in the prevalence of ECG abnormalities had been reported in several previous studies.

In addition, the limited studies that provide information on the variety of ECG changes prevalence in the local CKD population can make the physician less aware of the complications of CVD in late-stage CKD. The prevalence of ECG changes in local CKD patients in Indonesia may also be different from the other studies and literature that has been reported. The aim of this study was to scrutinize the ECG abnormalities among nondialysis late-stage CKD patients.

Materials and methods

This was a descriptive observational study using cross-sectional design, conducted at Dr. Soetomo General Hospital, Surabaya, Indonesia. This hospital is one of the biggest hospitals in Indonesia and also being functioned as referral hospitals for East Indonesia region. The study was conducted from 1 January to 31 December 2019. All adult patients with late-stage CKD (stage 3B, 4 and 5) that was hospitalized during the study period and consulted at department of cardiology and vascular medicine during their initial admission at emergency room were included in this study. Patients with previously hemodialysis, unstable hemodynamic, history of cardiac arrest, previously consumed antiarrhythmic drugs and lack of complete medical record data were excluded.

All patients gave their informed consent prior to their inclusion in this study. Information for informed consent was given before the participants signed the informed consent. Details that might disclose the identity of the participants were omitted. This study followed the principles of the Declaration of Helsinki and had been approved by the ethical committees of Dr. Soetomo General Hospital (Ethical clearance number: 1811/KEPK/II/2020).

All patients had resting 12-lead ECG examination. All ECGs were reviewed by qualified cardiologist for the following parameters: heart rhythm, heart rate, frontal axis, P wave, PR interval, QRS duration, pathological Q wave, fragmented QRS morphology, ST-segment deviation, QT interval and T wave abnormality. The aforementioned parameters were studied and measured manually and compared with published normal values. Normal limit and definition used for this study were shown in Table 1 and Table 2.

All statistical analyses were performed using SPSS 25.0 (IBM Corp., Armonk, N.Y., USA). For descriptive analysis, continuous data were given as mean $\pm$ SD or median (IQR) based on data distribution, while categorical data were given as n (%). One-Sample Kolmogorov-Smirnov was used to assess the data distribution.

Table 1: Normal limit for electrocardiographic parameters.

Parameters	Normal limit [37]
Rhythm	Sinus rhythm (Positive P wave in I, II, aVF and no AV dissociation)
Heart rate	60–100 bpm
Frontal axis	–30 to +90°
P Wave	<120 ms in duration and <2.5 mm in amplitude
PR Interval	120–200 ms
QRS Complex	< 120 ms and no fragmented QRS
Q Wave	Depth < 1/4 amplitude deflection of R wave and $\leq$ 40 ms
QT Interval	Corrected QT interval by Bazett's formula 330–440 ms in male or 330–450 in female
ST-Segment	Deviation at J-point < 1 mm
T Wave	No inverted T wave except in lead III, aVR and V1

bpm, beats per minute; mm, millimeter; ms, millisecond.

Results

There were 191 patients included in this study. Mean age was 52.2 ± 11.8 years old. Total number of male and female was almost equal. Most patients were in CKD stage 5. Mean creatinine serum was 10.5 ± 8.0 mg/dL and mean eGFR was 9.6 ± 4.4 mL/min/1.73 m². There were 143 (74.9%) patients with anemia (mean hemoglobin concentration was 8.1 ± 2.3 g/dL), 111 (58.1%) patients with HTN (Mean systolic blood pressure was 142.1 ± 28.2 mmHg and mean diastolic blood pressure was 83.7 ± 16.3 mmHg) and 75 (39.3%) with type 2 diabetes mellitus. Mean serum potassium of the patients in this study was 4.8 ± 1.06 mEq/L. There were only 5.8% patients with very high serum potassium level (>6.5 mEq/L) (Table 3).

Resting 12-lead ECG examination showed that the mean heart rate was 92.2 ± 15.6 beats per minute, mean PR interval was 157.3 ± 32.2 ms (ms), mean QRS duration 87.9 ± 17.9 ms and mean QTc interval was 442.9 ± 37.6 ms. At least one ECG abnormalities was found in 176 (92.1%) patients. The most common ECG abnormalities found were prolonged QTc interval (36.6%), fragmented QRS complex (fQRS) (29.8%), poor R wave progression (PRWP) (24.6%), peaked T wave (22.0%) and left ventricular hypertrophy (LVH) (16.7%) (Table 4).

Discussion

From previous epidemiological studies, patients with CKD are more susceptible in developing CVD. CVD can kill patients with CKD more often and more prematurely. The CVD spectrum in CKD and ESRD includes ischemic

Table 2: Definition used to determine ECG abnormality.

ECG Abnormality	Definition Used
FQRS complex	Additional spikes within the QRS complex [38]
LBBB	<ol style="list-style-type: none"> 1) QRS duration ≥ 120 ms, 2) Broad notched or slurred R wave in leads I, aVL, V5 and V6 and an occasional RS pattern in V5 and V6 attributed to displaced transition of QRS complex, 3) Absent Q waves in leads I, V5 and V6 or a narrow Q wave in the absence of myocardial pathology in the lead aVL, 4) R peak time greater than 60 ms in leads V5 and V6 but normal in leads V1, V2 and V3, when small initial R waves can be discerned in the above leads, 5) ST and T waves opposite in direction to QRS, 6) Positive T wave in leads with upright QRS, 7) Depressed ST segment and/or negative T wave in leads with negative QRS [39]
RBBB	<ol style="list-style-type: none"> 1) QRS duration ≥ 120 ms, 2) rsr', rsR' or rSR' in leads V1 or V2 where the R' or r' deflection is wider than the initial R wave [39]
Poor R wave progression	R wave in V3 or V4 ≤ 2 mm [40]
LAE	Any one of the following: <ol style="list-style-type: none"> 1) P wave in any lead > 0.11s, 2) Notched P wave with interpeak duration > 0.04s, 3) P wave axis $< 30^\circ$, 4) Area of negative P terminal force in lead V1 > 0.04 s mm, or 5) Positive P terminal force in lead aVL > 0.5 mm [41]
LVH	The sum of the S wave amplitude in lead V1 and the largest amplitude of R wave in either lead V5 or V6 ≥ 35 mm [42]
RAE	Any one of the following: <ol style="list-style-type: none"> 1) P wave in inferior leads II, III, aVF > 2.5 mm or 2) Positive P wave in V1 > 1.5 mm [41]
RVH	Any one of the following: <ol style="list-style-type: none"> 1) Tall R V1 > 6 mV, 2) Increased R:S ratio V1 > 1.0, 3) Deep S V5 > 10 mV, 4) Deep S V6 > 3 mV, 5) Small S V1 < 2 mV, 6) Small R V5,6 < 3 mV, 7) Reduced R:S ratio V5 < 0.75, 8) Reduced R:S ratio V6 < 0.4, or 9) Presence of QR V1 [43]
ST-Elevation	Two contiguous leads with ST-segment elevation ≥ 2.5 mm in men < 40 years, ≥ 2 mm in men ≥ 40 years, or ≥ 1.5 mm in women in leads V2–V3 and/or ≥ 1 mm in the other leads in the absence of LVH or LBBB [44]
ST-Depression	Depression of the ST-segment of ≥ 0.05 mV below the isoelectric line in leads V2 and V3 and ≥ 0.1 mV in the other lead [45]
Inverted T wave	Inverted T wave deeper than 1 mm [45]
Non-specific ST-T changes	<ol style="list-style-type: none"> 1) No ST-T depression ≥ 0.5 mm but ST-segment down-sloping and ST-segment or T-wave nadir at least 0.5 mm below the P-R baseline in any of leads I, II, aVL, or V2 to V6 2) ST-T depression ≥ 1.0 mm and ST-segment up-sloping or U-shaped in any of leads I, II, aVL, or V1 to V6 3) T-wave amplitude zero (flat), negative, or biphasic (negative–positive type only) with < 1.0 mm negative phase in leads I, II, aVL, or V3 to V6 when R-wave amplitude is ≥ 5.0 mm, 4) T-wave amplitude positive and T- to R-wave amplitude ratio of $< 1:20$ in any of leads I, II, aVL, or V3 to V6 when R wave amplitude in the corresponding leads was ≥ 10.0 mm [46]
Pathologic Q wave	Any Q wave with more than 40 ms width or a depth more than one-third of the adjacent R wave in more than two adjacent leads [47]
Peaked T wave	Subjective assessment of peaked T wave morphology (narrow, symmetric and tall T wave or T/R ratio ≥ 0.75) [48]
Low voltage	Peak-to-peak QRS voltage < 5 mm in all limb leads and < 10 mm in all precordial leads [49]

fQRS, fragmented QRS; LAE, left atrial enlargement; LBBB, left bundle branch block; LVH, left ventricular hypertrophy; mm, millimeter; ms, millisecond; mV, millivolt; RAE, right atrial enlargement; RBBB, right bundle branch block; RVH, right ventricular hypertrophy.

Table 3: Baseline characteristic of study population.

Variables	N=191 n (%)
Sex	
Male	95 (49.7)
Female	96 (50.3)
Age group (in years)	
≤ 30	10 (5.2)
31–40	25 (13.1)
41–50	37 (19.4)
51–60	77 (40.3)
61–70	33 (17.3)
> 70	9 (4.7)
Comorbidities	
Anemia	143 (74.9)
Hypertension	111 (58.1)
Type 2 Diabetes Mellitus	75 (39.3)
Metabolic Acidosis	63 (33.0)
Pneumonia	48 (25.1)
Hypoalbuminemia	48 (25.1)
Hyponatremia	38 (19.9)
Pleural Effusion	30 (15.7)
Pulmonary Edema	29 (15.2)
Nephrolithiasis	14 (7.3)
Pericardial Effusion	1 (0.5)
CKD Staging	
Stage 3B (GFR 30–44 mL/min/1.73 m ²)	8 (4.2)
Stage 4 (GFR 15–29 mL/min/1.73 m ²)	23 (12.0)
Stage 5 (GFR < 15 mL/min/1.73 m ²)	160 (83.8)
Potassium serum level (mEq/L)	
Hypokalemia (<3.5 mEq/L)	16 (8.4)
Normokalemia (3.5–5.0 mEq/L)	88 (46.1)
Hyperkalemia (> 5.0–6.5 mEq/L)	75 (39.3)
Severe hyperkalemia (> 6.5 mEq/L)	11 (5.8)

CKD, chronic kidney disease; GFR, glomerular filtration rate; mEq/L, milliequivalent per Liter.

heart disease, congestive heart failure, arrhythmias and peripheral vascular disease. Initially, it was estimated that this only occurred in the ESRD population which was 20–30 times more likely to die from CVD compared to the general population. However, this increased risk is spread across the CKD spectrum at all stages of CKD [10].

Anemia and hypertension were the most common comorbidities in our study and may become the main contributory factors in ECG abnormality. Anemia was found in 74.9% of our study population. Low levels of hemoglobin are associated with LVH, increasing of hospital admission rate and downgraded quality of life [11]. Hypertension existed in 58.1% of our subjects. The prevalence of HTN in CKD population depends on the stage of CKD and its cause. The pathophysiology of HTN in developing CKD includes salt retention, volume overload, sympathetic overactivity, endothelial dysfunction and blood pressure-associated hormonal disturbance.

Table 4: Electrocardiographic Abnormalities.

Abnormalities	N=191 n (%)
Ischemia	
Fragmented QRS complex	57 (29.8)
Pathological Q wave	26 (13.6)
Inverted T wave	14 (7.3)
ST Segment depression	13 (6.8)
ST Segment elevation	2 (1.0)
Chamber dilatation	
Left atrial enlargement	46 (24.0)
Left ventricular hypertrophy	32 (16.7)
Right atrial enlargement	2 (1.0)
Right ventricular hypertrophy	5 (2.6)
Electrolyte imbalance	
Prolonged QT interval	70 (36.6)
Peaked T wave	42 (22.0)
Blocks	
1st Degree atrioventricular block	14 (7.3)
Right bundle branch block	11 (5.8)
Wide QRS complex	7 (3.7)
Left bundle branch block	1 (0.5)
2nd Degree atrioventricular block	1 (0.5)
Arrhythmia	
Premature ventricular contraction	5 (2.6)
Atrial fibrillation	4 (2.1)
Premature atrial contraction	1 (0.5)
Others	
Poor precordial R wave progression	47 (24.6)
Non-specific ST-T changes	24 (12.6)
Frontal axis deviation	21 (11.0)
Low voltage	3 (1.6)
Short QT Interval	1 (0.5)

Uncontrolled HTN is also correlated with greater risk for cardiovascular (CV) morbidity and mortality in CKD [12].

Our study showed that abnormal ECGs were found in 92.1% of all CKD patients. This is higher than previous studies, where abnormal ECG measurements were found in 50–86% of all patients [4, 6, 11, 13, 14]. In this study, prolonged QTc interval was the most common ECG finding observed. Study by Yadla and Poosa showed that prolonged QTc was found in 13.3% of CKD population [13]. Nie et al. showed that 65% of patient undergoing hemodialysis (HD) had prolonged QTc interval [15]. These ECG markers are also useful to detect silent CV disease in the CKD setting [16]. This result also found in Bignotto et al.; approximately half of the dialysis studied population had prolonged QTc [6]. Prolonged QTc is reported to be very common in patients on HD and the factors associated with the same were found to be abnormal levels of serum calcium, phosphorus, potassium, ferritin, uric acid and BNP. Recent studies showed the significant association between interval of QTc and sudden cardiac death in

hypertrophic cardiomyopathy and vascular calcification, helped us to recognize the essentials of QTc interval as risk evaluation in patients undergoing dialysis for SCD event. QTc interval presents more frequently as the stage of CKD progressing, but in dialysis patients, QTc interval was not significantly dissimilar as compared to nondialysis ESRD patients [17]. The phenomenon of higher serum creatinine correlate with long QTc interval could be explained by numerous factors influencing the QT interval: 1) Patients with CKD often take many medications that can cause QT prolongation; 2) Secondary hyperparathyroidism, a common complication of CKD, could lead to hypocalcemia and hypomagnesemia; 3) Renal anemia, anisocytosis and macrocytosis are also associated to long QT due to subsequent hypoxia, autonomic dysfunction and decreased myocardial oxygen supply; 4) Disturbance of delayed rectifier potassium channels; 5) Hypertension can significantly reduce potassium current densities (I_{peak}, I_{to}, I_{Kur}, I_{ss} and I_{k1}) and increase the L-type calcium channel and 6) Reactive oxygen species and uremic toxin leading to inflammatory condition and oxidative stress itself may also predispose to cardiac electrical remodeling [18, 19]. Therefore, the prolongation of the QTc interval is common in hospitalized patients with CKD.

fQRS became the second most common abnormality found in our study. It was a marker of heterogeneous depolarization of ventricular myocardium caused by myocardial fibrosis and myocardial scarring. Cardiac remodeling is one of the significant myocardial structural abnormality in CKD patients [20]. While prevalence of fQRS is uncommon in population of previous study. Terho et al. found fQRS in 9.2% of Finnish middle-aged healthy subjects [21]. Adar et al. evaluated the frequency and association of fQRS with asymptomatic left ventricle (LV) dysfunction in individuals with CKD. In general, the prevalence of fQRS was about 60% among CKD patients with preserved LV ejection fraction. In addition, fQRS was independently correlated with asymptomatic LV dysfunction in CKD patients [22]. The previous study by Liu et al. mentioned that fQRS prevalence was 30.32% (94 of 310 patients). The distribution of fQRS in CKD Stage 3, 4 and 5 patients were 30.77, 20.00 and 32.03%, respectively. In the inferior leads, fQRS was more frequent (53.19%), compared to anterior (21.28%) and the lateral leads (25.53%). However, lateral leads fQRS was strongly associated with a lower LV ejection fraction [23]. Our result is consistent with Adar et al. and Liu et al., which showed that fQRS is one of the common findings among our CKD patients (29.8%). In contrast with Liu

et al., our study did not specify the location of fQRS. Liu et al. also stated that anterior and inferior fQRS were not related with an escalating risk of morbidity and mortality even in individuals with a previously known cardiac disease. On the contrary, lateral fQRS predicted, arrhythmic, cardiac and all-cause death in those individuals [23].

PRWP was frequently found in 24.6% of our population. Previous studies by Yadla and Poosa also showed that PRWP was common in patients on maintenance HD (11.6%) [13]. Zema and Kligfield identified that PRWP might be caused by previous anterior acute myocardial infarction, LVH, type C right ventricular hypertrophy (RVH) or may exist in otherwise healthy subjects [24].

Patients with CKD, especially in the advanced stage, are at a higher risk for incident of CVD complication and sudden cardiac death than the general population. Previous population-based research including 18,864 samples in 2012, shown silent MI (SMI) increased with decreasing eGFR from 4% in those with eGFR ≥ 60 mL/min/1.73 m² to 13% with eGFR < 30 mL/min/1.73 m² [25]. In patients with CKD, SMI was independently associated with worse outcomes during follow-up [26]. In previous study by Shafi et al. [4], pathologic Q waves and deviation of ST-segment were found in 23.4 and 27.2%, respectively; and these were similar to our study: frequency of Q waves, ST-segment elevation and ST-segment depression were 13.6, 1.0 and 6.8%, respectively. Inverted T wave which may become one of ECG signs of CAD was also found in 7.3% in our CKD patient.

Hyperkalemia is associated with progressing ECG abnormalities starting from tall and peaked T waves, reduced QT interval, followed by prolonging of the PR interval and widening QRS duration, disappearing of the P wave, appearing idioventricular rhythms and significant broadening of the QRS up to a sine-wave pattern. In our study, hyperkalemia is found in 45.5% population. Peaked T wave as one of the most common ECG changes caused by hyperkalemia was found in 22% population in our study. While several other ECG abnormalities that may be caused by hyperkalemia in our study include 1st Degree trioventricular block (AV block) (7.3%), wide QRS complex (3.7%) and short QTc interval (0.5%). Activation of the potassium channels can accelerate membrane repolarization, then overall membrane depolarized which inactivates sodium channels [27]. Accelerated cardiac action potential repolarization causes T waves tenting.

Recent studies support the hypothesis of association between systolic dysfunction and LVH regression in baseline and one-year dialysis ECG of CKD patients with a

lower risk of new-onset heart failure [11]. In our study, the ECG marker associated with chamber dilatation found are left atrial enlargement (LAE) (24%), LVH (16.7%), RVH (2.6%) and right atrial enlargement (1%). The prevalence of LVH in ESRD is vary, depending on the characteristic of the study population. Previous studies showed that the prevalence of LVH are around 40% [8, 11], but other study showed that the prevalence was 80% [28]. The causes of CKD seem to have contributed on the ECG abnormality as prevalence of LVH and LAE were greatest among, diabetic nephropathy, hypertensive renal disease and chronic glomerulonephritis [17].

Non-specific ST-T changes were found in 12.6% of the population. This result is lower than previous studies presented nonspecific ST-T changes in 30% of cases [29]. Badheka et al. stated that nonspecific ST-T changes on ECG were correlated with a greater prevalence of CVD risk factors in the common population. The further investigation suggests that clinical events can be preceded by depolarization and repolarization abnormalities, while isolated nonspecific ST-T changes on ECG were associated with increasing risk of cardiovascular morbidity and all-cause mortality [30].

The importance of determining the electrical axis is essential in the identification of subtle early caution indicators of any cardiovascular alterations of the patient [31]. Furthermore, previous research by Rodriguez et al. showed that the QRS frontal axis was more leftward in the population with significant microalbuminuria [32]. Sachdeva et al. also showed left axis deviation in 15% of their CKD patients [14]. Our study showed the frequency of frontal axis deviation was found in 11% CKD patients without further investigation of the microalbuminuria status. In contrast with previous studies, Korkmaz et al. found changes in the QRS axis are rarely emphasized in such patients and no significant differences was seen in the QRS axis in HD patients with or without CVD [33].

Cohort studies by Flueckiger et al. found nearly 10% of the ESRD population evaluated for renal transplantation had a prolonged PR interval. Risk of death was twice as high for patients with PR > 200 ms compare to patients with PR ≤ 200 ms [34]. As mentioned in the previous study conducted by Mandic et al., among 140 patients with routine HD, first degree AV block was found in 2.9% patients, but there were no second- and third-degree AV blocks [35]. These findings were consistent with our result that showed 7.3% CKD patients with first degree AV Block and only 0.5% patient with second-degree AV block.

Other arrhythmias including conduction abnormalities were found in our study were right bundle branch

block (RBBB) (5.8%) and left bundle branch block (LBBB) (0.5%). Our results were lower than the recent research by Bignotto et al. which showed the prevalence of RBBB and LBBB were 10.05 and 15.08%, respectively [6]. Shafi et al. also found a higher prevalence of RBBB as 12.8% than LBBB as 9.6% [4]. Premature ventricular contraction (PVC) and premature atrial contraction (PAC) were also found in our study with prevalence of 2.6 and 0.5%, respectively. Yadla showed PVC prevalence of 6%, while Shafi et al. showed PVC prevalence of 15.2% and PAC 4.8% [4, 13]. Atrial fibrillation (AF) (2.1%) was the most common non-sinus rhythm found in our study, while the rest was junctional rhythm (0.5%). Previous study by Bignotto et al. also showed AF was the most common nonsinus rhythm (4.44%) in chronic HD patients [6]. CKD patients at all stages may have a broad spectrum of cardiac arrhythmias, including AF, supraventricular tachycardias, PVC, sustained “malignant” ventricular tachyarrhythmias and SCD. In later stages, bradyarrhythmias and asystole associated with hyperkalemia or another electrolyte imbalance may also present. Pathophysiology of arrhythmias in patients with CKD is related to many changes in cardiac or noncardiac factors in CKD, such as: LVH, fibrosis, autonomic imbalance, rapid fluid and electrolyte shifts [36].

There were several limitations in our study. First, further risk factors for ECG abnormalities were not taken into account for the analysis. Second, this was a cross-sectional study, single center with small sample sizes in once measurement. Third, necessary information such as echocardiography or chest X-ray may be unavailable. The strength of this study was data collected actually, prevalence estimation could be done consecutively, complete study populations makes selection bias minimally and collect data process independently.

Conclusions

ECG abnormalities are common among nondialysis late-stage CKD patients. The most common features of resting ECG patterns observed in those patients were prolonged QTc interval and fragmented QRS. Poor Precordial R Wave Progression, Peaked T wave and left ventricular hypertrophy were also common patterns in nondialysis late-stage CKD patients. Given the fact that long-term CKD influences the pathogenesis of cardiorenal syndromes, cardiovascular risk factors, cardiovascular diseases and substantial cardiovascular mortality, there is a need to screen Indonesian CKD patients who are at risks of getting earlier complications.

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Ethical approval: The research related to human use has complied with all the relevant national regulations, institutional policies and in accordance with the tenets of the Helsinki Declaration, and has been approved by the authors' Institutional Review Board (Ethical clearance number: 1811/KEPK/II/2020).

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