Diagnostic value of electrocardiogram in cardiac tamponade

Kai Ping Ang, MRCP[1], Rusli Bin Nordin, PhD[2], Stanley Chin-Yu Lim, MRCP[1], Chuey Yan Lee, MRCP[1], Hou Tee Lu, FRCP[1,2]

[1]Department of Cardiology, Sultanah Aminah Hospital, Johor Bahru, Johor, Malaysia, [2]Jeffrey Cheah School of Medicine and Health Sciences, Monash University Malaysia, Johor Bahru, Johor, Malaysia

ABSTRACT

Introduction: We aim to study the diagnostic value of electrocardiogram (ECG) in cardiac tamponade.

Methods: This study was a single centre, retrospective case-control study. We recruited 42 patients diagnosed with cardiac tamponade of various aetiologies confirmed by transthoracic echocardiography and 100 controls between January 2011 and December 2015. The ECG criteria of cardiac tamponade we adopted was as follows: 1) Low QRS voltage in a) the limb leads alone, b) in the precordial leads alone or, c) in all leads, 2) PR segment depression, 3) Electrical alternans, and 4) Sinus tachycardia.

Results: Malignancy was the most common causes of cardiac tamponade, the two groups were of similar proportion of gender and ethnicity. We calculated the sensitivity (SN), specificity (SP), positive predictive value (PPV), and negative predictive value (NPV) of each ECG criteria. Among the ECG abnormalities, we noted the SN of 'low voltage in all chest leads' (69%), 'low voltage in all limb leads' (67%) and 'sinus tachycardia' (69%) were higher as compared to 'PR depression' (12%) and 'electrical alternan' (5%). On the other hand, 'low voltage in all chest leads' (98%), 'low voltage in all leads' (99%), 'PR depression' (100%) and 'electrical alternans' (100%) has highest SP.

Conclusion: Our study reaffirmed the findings of previous studies that electrocardiography cannot be used as a screening tool for diagnosing cardiac tamponade due to its low sensitivity. However, with clinical correlation, electrocardiography is a valuable adjuvant test to 'rule in' cardiac tamponade because of its high specificity.

KEY WORDS:
Cardiac tamponade, electrocardiogram, cardiac diagnostic test, sensitivity, specificity

INTRODUCTION

Pericardial effusion is an abnormal accumulation of fluid in the pericardial cavity. Cardiac tamponade occurs when a large amount of fluid accumulates in the pericardium, leading to hemodynamic compromise and death if not treated timely.[1] Early diagnosis is crucial in the management of pericardial tamponade. Patients with pericardial effusion may quickly progress to cardiac tamponade requiring urgent pericardiocentesis. Although cardiac tamponade is considered a clinical diagnosis, clinical findings like dyspnoea, hypotension, tachycardia, elevated jugular venous pressure, and pulsus paradoxus, are known to have limited sensitivity and specificity.[2] A systemic review by Roy et al. found that dyspnoea (sensitivity range, 87-89%), tachycardia (pooled sensitivity, 77%), pulsus paradoxus (pooled sensitivity, 82%), elevated jugular venous pressure (pooled sensitivity, 76%), and cardiomegaly on chest radiograph (pooled sensitivity, 89%) occur in majority of patients with cardiac tamponade.[3] Imaging techniques such as echocardiography or ultrasound scan can accurately detect and quantify the size of pericardial effusions and is a gold standard diagnostic tool for the diagnosis of cardiac tamponade.[4] Nevertheless, these techniques are often not feasible as screening tests for pericardial effusion at frontline physician such as general practitioners or primary care community health clinics. Furthermore, echocardiography requires a trained sonographer or cardiologist for interpretation. It is may not be practical and could be costly to send all patients for universal screening by echocardiography to rule out cardiac tamponade based on clinical suspicion alone. In contrast, 12-lead electrocardiography (ECG) is a non-invasive, cost effective, easy to perform, and readily available diagnostic tool in most of the health care settings. We will provide a brief background on the use of the 12-lead ECG for diagnosing cardiac tamponade. Spodick et al., had made a pivotal contribution to the research of ECG abnormalities in pericarditis.[1,2,5,6] In 1996, Eisenberg et al. applied a set of ECG criteria (low QRS amplitude, PR depression and electrical alternans) to demonstrate that low voltage and PR depression were suggestive but not diagnostic of cardiac tamponade.[7] Another researcher found that low QRS voltage was observed in the majority of patients with cardiac tamponade but not in patients with pericarditis and large pericardial effusion.[8] Other studies showed that ECG abnormalities (low voltage, electrical alternans and tachycardia) rule in cardiac tamponade better than they rule it out.[9] In a recent study, the presence of low QRS voltage, PR segment depression and electrical alternans in combination were useful as a supplementary diagnostic tool of pericardial tamponade.[10] We aim to build on the previous work by expanding the evidence in order to improve generalizability of the usage of ECG in the diagnosis of cardiac tamponade.

This article was accepted: 20 May 2018
Corresponding Author: Hou Tee Lu
Email: luhoutee@gmail.com; lu.hou.tee@monash.edu

MATERIALS AND METHODS

This study was a single centre, retrospective case-control study conducted in Sultanah Aminah Hospital, a tertiary care-hospital in the discipline of cardiology the southern region of peninsular Malaysia. In this study, we retrospectively identified patients admitted to cardiology and medical units between January 2011 and December 2015 with a primary diagnosis of cardiac tamponade. We focused on a population diagnosed with cardiac tamponade of various aetiologies and a group of normal patients (controls).

Cardiac tamponade

Patients 18 years and above with cardiac tamponade requiring hospitalization and pericardiocentesis were recruited. Pericardial effusion is defined as abnormal accumulation of fluid in the pericardial cavity. Cardiac tamponade is defined as a life-threatening condition due to accumulation of large pericardial effusion resulting in compression of the heart, circulatory collapse or cardiogenic shock.[1,4] Cardiac tamponade is confirmed by transthoracic echocardiography (TTE) (Philips iE33, Philips Medical Systems, Andover, MA, USA) with at least one the following features: 1. Diastolic collapse of the free walls of right atrium, 2. Right ventricular compression in diastole, and 3. Exaggerated respiratory variation in inflow velocity: an inspiratory decrease in peak mitral inflow velocity > 30%, and an inspiratory increase of peak tricuspid inflow velocity > 50%.[4] Eligible cases were patients with a confirmed diagnosis of cardiac tamponade based on a documented admission of TTE. A standard 12-lead ECG at a paper speed of 25 mm/s and an amplification of 10 mm/mV was performed admission. All patients diagnosed with cardiac tamponade were admitted to the coronary care unit and reviewed by a cardiologist.

Controls

The eligible controls (normal subjects or patients with normal echocardiography) were selected as reference standard. Similar to study cases, the controls were identified from daily admissions from the same hospital. All controls had TTE studies. Most of the control patients were diagnosed with coronary artery disease and congestive heart failure but none of them had pericardial effusion demonstrated by TTE during the same admission. For each case of cardiac tamponade, we enrolled two controls during the same period of admission (within a month). Collaboration was sought with cardiologists, general physicians and nurses. They were actively involved in the identification of eligible patients, ECG diagnosis and review of patient's medications. The final ECG interpretation of every patient was independently evaluated by two cardiologists.

Data such as demographic characteristics (age, gender, ethnicity), co-morbidities (cigarette smoking, diabetes mellitus, hypertension), admission vital signs (heart rate, systolic and diastolic blood pressure), ECG findings, laboratory results on admission (fasting blood glucose, urea, creatinine, alanine aminotransferase, estimated glomerular filtration rate [eGFR] and total cholesterol) and outcomes of cardiac tamponade (in-hospital death) were extracted from the patients' records using a standardized data collection form. In order to minimize selection bias, we checked each patient's identity and reference number to avoid the same patient being included twice. Only the data on first admission was recorded. We excluded patients with incomplete information on the demographic characteristics, laboratory results and ECG. Post-myocardial infarction pericarditis and trauma patients were excluded from the analysis.

ECG criteria

We adopted the ECG criteria for the diagnosis of cardiac tamponade based on an earlier study by Eisenberg et al.[7] Indeed, similar criteria were used by other ECG diagnostic studies for cardiac tamponade.[8-10] Low voltage definition included patients who had 1) Low voltage in the limb leads alone: defined as a QRS amplitude less than 5 mm in all limb leads, 2) Low voltage in the precordial leads alone: defined as a QRS amplitude less than 10 mm in all precordial leads, and 3) Low voltage in all leads: defined as a QRS amplitude less than 5 mm in all limb leads plus a QRS voltage less than 10 mm in all precordial leads. PR segment depression was defined as a depression 1 mm or greater of the PR segment in at least one lead other than lead aVR. Electrical alternans was defined as a peak-to-peak QRS amplitude change of 1 mm or greater in each successive beat in at least one lead. Sinus tachycardia is defined as heart rate of the admission ECG exceeding 100 beats per minute.

Statistical Analysis

We assessed the differences between the baseline characteristics, vital signs at presentation, laboratory results and ECG findings of patients with cardiac tamponade and control. Numerical data was recorded as mean and standard deviation for normally distributed data, and median and interquartile range for non-normally distributed data. Categorical data was expressed as frequencies and percentages. A chi square test was used to assess differences between categorical variables; independent student's t-test (parametric analysis) or Mann-Whitney U test (non-parametric analysis- if the assumption of distributional normality was significantly violated) was used to test differences between numerical variables. We calculated the sensitivity (SN), specificity (SP), positive predictive value (PPV), and negative predictive value (NPV) of each ECG criteria using MedCalc Trial Version 16.8.4. All other statistical calculations were performed using the SPSS statistics software (version 20, IBM, Armonk, New York).

Ethics approval

Approval for this study was granted by the Ethics Committee (National Medical Research Register [NMRR]) (Medical Research Ethics Committee Approval code: NMRR-15-2123-24072 - [Research ID: 24072]). Written consent was waived by the Ethics Committee.

RESULTS

A total of 50 patients with cardiac tamponade (confirmed by echocardiography) and 100 patients without cardiac tamponade as controls (with a ratio of 1:2) were recruited during the study period. Among the patients diagnosed with cardiac tamponade, only 42 patients fulfilled the inclusion criteria. Eight patients with cardiac tamponade were

Table I: Baseline demographic characteristics, vital signs and laboratory results

Variables	Cardiac tamponade				P value
	Present (n=42)		Absent (n= 100)		
Age¶, (year)	46.8	(17.3)	54.5	(14.9)	0.009§
Male (n, %)	30	(71.4)	67	(67.0)	0.605¥
Ethnicity (n, %)					
Malay	21	(50.0)	53	(53.0)	0.744¥
Non-Malay (Chinese, Indian or others$^{\eta}$)	21	(50.0)	47	(47.0)	
Smoking status (Current / Former) (n, %)	14	(35.9)	14	(14.0)	0.004¥
Hypertension (n, %)	10	(23.8)	43	(43.0)	0.031¥
Diabetes mellitus (n,%)	10	(23.8)	30	(30.0)	0.454¥
Vital signs on admission					
Heart rate‡, (beat/min)	107	(96, 120)	74	(62, 93)	0.001#
Systolic blood pressure‡, (mmHg)	117	(101,131)	133	(120,149)	0.006#
Diastolic blood pressure¶, (mmHg)	76	(14)	79	(14)	0.316§
Laboratory results					
Hemoglobin‡, g/dl	10.6	(9.4,12.4)	13.3	(10.7,15.2)	0.003#
Alanine aminotransferase‡, u/l	22	(12,38)	21	(13,40)	0.851#
Creatinine‡, µmol/l	80	(55,119)	90	(72,125)	0.493#
eGFR¶, ml/min/1.73m^2	104	(89)	76	(36)	0.018§

All categorical variables are expressed as number and percentage (n, %). eGFR, estimated glomerular filtration rate calculated by the abbreviated MDRD equation
$^{\eta}$Indigenous (Orang Asli) and other non-Malaysians
‡Median (interquartile range)
¶Mean (standard deviation)
¥ Pearson Chi square value, χ^2 test for 2x2 table (using Fisher-Exact test) for categorical variables.
§Independent student T test
#Mann Whitney U test

Table II: 12-lead ECG abnormalities among patients with cardiac tamponade and controls

ECG abnormalities	Cardiac tamponade (n=42)		Control (n=100)	
	Present	Absent	Present	Absent
Low voltage				
All limb leads	28	14	5	95
All chest leads	29	13	2	98
All leads (limb + chest leads)	27	15	1	98
PR segment depression	5	37	0	100
Electrical alternans	2	40	0	100
Sinus tachycardia	29	13	20	80

Table III: Diagnostic value of ECG in predicting cardiac tamponade

ECG signs	SN (%)	95% CI	SP (%)	95% CI	PPV (%)	95% CI	NPV (%)	95% CI
Low voltage in all limb leads	67	(50-79)	95	(88-98)	85	(67-94)	87	(79-92)
Low voltage in all chest leads	69	(53-82)	98	(92-99)	94	(77-99)	88	(80-93)
Low voltage in all leads	64	(48-78)	99	(94-99)	96	(80-99)	87	(79-92)
PR depression	12	(4-26)	100	(95-100)	100	(46-100)	73	(65-80)
Electrical alternans	5	(1-17)	100	(95-100)	100	(19-100)	71	(63-79)
Sinus tachycardia	69	(53-82)	80	(71-87)	59	(44-73)	86	(77-92)

SN, sensitivity, SP, specificity, CI, confidence interval (%); PPV, positive predictive value; NPV, negative predictive value

excluded from analysis because of incomplete information. Therefore, the analysis was performed on the remaining 142 patients. Among the 42 patients diagnosed with cardiac tamponade, malignancy (40.5%) was the most common cause, followed by infection (19.0%), renal insufficiency (21.4 %), autoimmune disease (2.4%) and idiopathic causes (16.7%) (Fig. 1).

Table I lists depicts the demographic characteristics, vital signs on admission and laboratory results of the study population. The mean age of the cardiac tamponade group was younger as compared to the control group (46.8 vs. 54.5 year, respectively, p=0.009). The two groups were of similar proportion for gender (p= 0.605) and ethnicity (p= 0.744). Patients with cardiac tamponade had a higher heart rate (107 vs. 74 beat/min, respectively, p=0.001) and lower systolic blood pressure (117 vs. 133 mmHg, respectively, p=0.006) at admission as compared to controls.

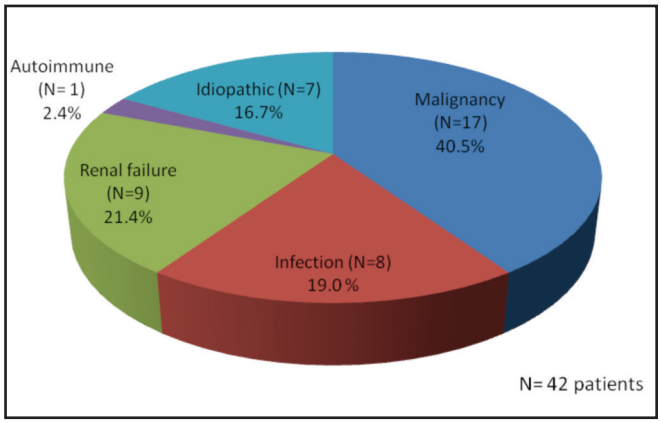

Fig. 1: Causes of cardiac tamponade.

Table II shows the presence or absence of ECG abnormalities among the patients with cardiac tamponade and controls. Based on this table, we calculated the SN, SP, PPV, and NPV of each ECG criteria.

Table III indicates the SN, SP, PPV, and NPV of each ECG abnormalities in predicting cardiac tamponade. Among the ECG abnormalities, 'low voltage in all chest leads' (69%; 95% confidence interval [CI]: 53-82%), 'low voltage in all limb leads' (67%; 95% CI: 50-79%) and 'sinus tachycardia' (69%; 95% CI: 53-82%) had higher SN as compared to 'PR depression' (12%; 95% CI: 4-26%) and 'electrical alternans' (5%; 95% CI: 1-17%), respectively. On the other hand, 'low voltage in all chest leads' (98%; 95% CI: 92-99%), 'low voltage in all leads' (99%; CI: 94-99%), 'PR depression' (100%; 95% CI: 95-100%) and 'electrical alternans' (100%; 95% CI: 95-100%) had highest SP. In our cohort, 'PR depression' and 'electrical alternans' had the greatest PPV (100%) but a NPV of 73% and 71%, respectively, for cardiac tamponade. The 'low voltage in all chest leads' had the greatest NPV (88%) but a PPV of 94%.

DISCUSSION

Several ECG abnormalities including low QRS voltage, PR segment depression, electrical alternans and sinus tachycardia have been associated with cardiac tamponade.[7-10] In the previous studies, the SN, SP, and predictive values of individual ECG abnormalities in estimating cardiac tamponade exhibited a wide range of values. One distinct feature in our study is that the ECG diagnostic values were derived from a population with cardiac tamponade and controls (normal subjects), and that was least evaluated in the previous studies. Most patient populations in the previous studies had high prevalence of pericardial effusions, and control subjects without effusions were often not included (except the study by Eisenberg et al. in which 19 controls were included in the analysis). Therefore,[7] results from the present study should be interpreted within the context of the overall design. However, despite the differences in the study populations and its designs, our results indicated that ECG abnormalities were generally specific but less sensitive in diagnosing cardiac tamponade. Overall, our study supports the findings of previous cross sectional studies that ECG abnormalities on its own are not reliable for diagnosing cardiac tamponade.[7-10] The following discussion will focus on each of the aforementioned ECG abnormalities applied in our study to diagnose cardiac tamponade.

Low voltage QRS complex

In agreement with previous retrospective and prospective studies,[7-10] low QRS voltage (chest leads, limbs leads and all leads) has high specificity but low sensitivity in diagnosing cardiac tamponade. In our study, 'low voltage of all leads' had the highest specificity (99%) as compared to 'low voltage in all chest leads' (98%) and 'low voltage in all limb leads' (95%) to diagnose cardiac tamponade. However, the sensitivity for 'low voltage of all leads' (64%), 'low voltage in all chest leads' (69%) and 'low voltage in all limb leads' (67%) were not good as compared to specificity. In the previous studies, the 'low voltage in all leads' criteria exhibited a sensitivity of 8 to 22% and a specificity ranging from 99 to100%.[7,9,10] An analysis from a series of early studies involving patients with cardiac tamponde from 1978 to 1996 by Roy et al. found that the pooled sensitivity of low QRS voltage was only 42% (95% CI, 32%-53%).[3] Low voltage is frequently caused by conditions that separate the electromotive wave conduction as a result of increased distance between the heart and the chest wall. Cardiac causes of low QRS voltage include multiple myocardial infarction, cardiomyopathies and hypovolemia because of cancellation and diminished electromotive force generation. Non-cardiac causes of low QRS voltage include pericardial effusion, tamponade, constrictive pericarditis, pneumopericardium, chronic obstructive airway disease, extensive skin burns and pneumothorax.[11] Meyer et al. has shown that the ECG findings of low QRS voltage was weakly correlated with effusion size in a group of patients with varying severity of pericardial effusion (mild, moderate and large) as compared to control subjects (without pericardial effusion).[12] However, Bruch et al. found that low QRS voltage is observed in the majority of subjects with cardiac tamponade but not in patients with pericarditis and large pericardial effusion suggesting a relationship between inflammation and low QRS voltage.[8]

PR segment depression

PR segment depression and ST junction deviations were known to be found in acute pericarditis. Eisenberg et al.[7] found that PR segment depression had a sensitivity of 42 % and a specificity of 86% in patients with cardiac tamponade. Kudaiberdiev et al.[13] found that 46% of patients with pericardial disease had signs of PR segment depression. In addition, PR segment depression was found to be associated with unfavorable outcome such as recurrent pericardial disease, heart failure and death as compared to patients without PR segment depression.[13] Our result showed that the majority of patients with cardiac tamponade did not have 'PR segment depression'. In our study, 'PR segment depression' had a sensitivity of only 12% but a specificity of 100% in diagnosing cardiac tamponade.

Electrical alternans

Consistent with the results of previous studies,[1,9,10] we found that the 'electrical alternans' is not always present in cardiac tamponade but it is very specific for this condition. The SN

and SP of 'electrical alternans' were similar to 'PR segment depression'. In other words, 'electrical alternans' was not very diagnostic for cardiac tamponade because the sensitivity was only 5% and a NPV of 71%. Electrical alternans is produced when the heart is oscillating within the pericardial sac distended by fluid and when associated with pericardial effusion is strongly suggestive of impending or established cardiac tamponade.[1,10] However, electrical alternans can be caused by a number of different clinical settings besides pericardial effusion and cardiac tamponade, including supraventricular and ventricular tachycardias, electrolyte abnormalities, drugs, hypothermia, prolonged QT syndromes, and bradycardia.[14]

Sinus tachycardia

Our results are consistent with previous publications[9,10] that sinus tachycardia (a heart rate of more than 100 beats per minute) has higher sensitivity but low specificity as compared to other ECG criteria for the diagnosis of cardiac tamponade. Sinus tachycardia commonly occur in cardiac tamponade as a compensatory mechanism.[15] Tachycardia is invariably present in cardiac tamponade except in patients with bradycardia during uremia and patients with hypothyroidism.[1] The finding of sinus tachycadia is clinically valuable as shown by Mathur et al. that the absence of tachycardia decreased the probability of tamponade by 50%. Meyers et al.[12] studied ECGs obtained before and after the development of a pericardial effusion found that the development of sinus tachycardia and reduction in QRS voltage were associated with the development of a pericardial effusion.[12]

Practical application of ECG in the diagnosis of cardiac tamponade

A perfect test has 100% sensitivity and 100% specificity. In real life, most clinical tests fall short to this ideal.[16] Sensitivity refers to how good a test is at correctly identifying people who have the disease. On the other hand, specificity is concerned with how good the test is at correctly identifying people who doesn't have the disease.[17] When a test has high specificity, a positive test 'rules in' the diagnosis; when a test has high sensitivity, a negative test 'rules out' the diagnosis. For clinicians, the terms PPV and NPV are preferred when considering the value of a test. PPV and NPV are dependent on the prevalence of the disease in the population of interest. For any diagnostic test, the PPV will fall as the prevalence of disease falls while the NPV will rise.[17] Knowing the ECG abnormalities were generally specific but less sensitive in diagnosing cardiac tamponade, it means that ECG is good in its PPV to 'rule in' cardiac tamponade but not useful as a screening tool. With this fundamental understanding and knowing the limitation of ECG (as illustrated from current and previous studies), the authors recommend first to identify the patients with clinical suspicion of cardiac tamponade, and then to correlate with the ECG abnormalities. Good history and physical examination are invaluable to identify patients with risk factors (a population with higher disease prevalence) and this will increase the PPV of ECG. For example, acute pericarditis, anticoagulant treatment, malignancy or chest trauma are risk factors of cardiac tamponade.[18] Signs of sinus tachycardia, pulsus paradoxus, cardiomegaly, clear lung field on chest radiograph[2,3] plus ECG abnormalities should quickly prompt a diagnosis of cardiac tamponade. In brief, when faced with a patient with risk factors and clinical signs of cardiac tamponade, ECG abnormalities may help guide decisions about the appropriateness of urgent echocardiography and management.

LIMITATIONS

A number of limitations should be noted. Firstly, our study involved a single centre with small sample size. Secondly, our patient population was restricted to hospitalized patients with cardiac tamponade. Hence, referral bias may occur as we may not able to capture those patients diagnosed with cardiac tamponade that was admitted elsewhere. Finally, our study was a retrospective case control study. We could only identify ECG abnormalities that were captured in our medical record. The appearance and/or disappearance of an ECG sign may be better identified by doing serial ECGs than a single ECG. Therefore, some ECG abnormalities may not be captured and may affect our calculations. Furthermore, despite our effort to minimize sampling bias, the clinical parameters differed between the cardiac tamponade and the control groups. Hence, we cannot be sure that the controls ideally represent the source population in which the cases belong, and these discrepancies might affect our result.

CONCLUSION

Our study reaffirmed the findings of previous studies that electrocardiography cannot be used as a screening tool for diagnosing cardiac tamponade due to its low sensitivity. However, with clinical correlation, electrocardiography is a valuable added test to 'rule in' cardiac tamponade because of its high specificity.

ACKNOWLEDGEMENTS

We would like to acknowledge Director General of Health of Malaysia (Ketua Pengarah Kesihatan) in giving the support for this research. We would also like to thank the assistant medical officers, technicians and nurses in Hospital Sultanah Aminah (Johor Bahru) for their invaluable help.

COMPETING INTEREST

The authors declare that they have no competing interests.

REFERENCES

1. Spodick DH. Acute cardiac tamponade. NEJM 2003; 349: 684-90.
2. Spodick DH. Acute cardiac tamponade. Pathologic physiology, diagnosis and management. Prog ress In Cardiovasc Dis 1967; 10:(1): 64-96.
3. Roy CL, Minor MA, Brookhart MA, Choudhry NKet al. Does this patient with a pericardialeffusionhavecardiactamponade?JAMA2007;297(16): 1810-18.
4. Jung HO. Pericardial effusion and pericardiocentesis: role of echocardiography. Korean Circ J 2012; 42: 725-34.
5. Spodick DH. Diagnostic electrocardiographic sequences in acute pericarditis. Significance of PR segment and PR vector changes. Circulation 1973; 48: 575-80.
6. Spodick DH. Risk prediction in pericarditis: whotokeep in hospital? Heart 2008; 94: 4.
7. Eisenberg MJ, Romeral LMD, Heidenreich PA, Schiller NB, Evans GT Jr.et al. The diagnosis of pericardial effusion and cardiac tamponade by 12-Lead ECG. A tTechnology aAssessment. Chest 1996; 110(2): 318-24.

8. Bruch C, Schmermund A, Dagres N, Bartel T, Caspari G, Sack S at al. Changes in QRS voltage in cardiac tamponade and pericardial effusion: reversibility after pericardiocentesis and after anti-inflammatory drug treatment. J Am Coll Cardiol 2001; 38(1): 219-26.
9. Mathur AP, Saini A, Lucas BP, AlYousef T, Margeta B, Mba B et al. Diagnostic accuracy retrospectively of electrocardiographic findings and cancer history for tamponade in patients determined to have pericardial effusion by transthoracic echocardiogram. Am J Cardiol. 2013; 111(7):1062-6.
10. Argula RG, Negi SI,Banchs J,Yusuf SWetal. Roleofa12-leadelectrocardiogramin the diagnosis of cardiac tamponade as diagnosed by transthoracic echocardiography in patients with malignant pericardial effusion. Clin Cardiol 2015; 38(3): 139-44.
11. Madias JE. Low QRS voltage and its causes. J of Electrocardiology 2008; 41(6): 498-500.
12. Meyers DG, Bagin RG and Levene JF. Electrographic changes in pericardial effusion. Chest 1993; 104(5): 1422-6.
13. Kudaiberdiev T, Dzhumagulova A, Joshibayev S, Tilemanbetova K, Imanalieva Ge et al. Electrocardiographic abnormalities in patients with pericardial disease — Association of PR segment depression with arrhythmias and clinical signs: Experience of cardiac surgery center. J of Electrocardiology 2016; 49(1): 29-36.
14. Chung KY, Walsh TJ, Massie E. Electrical alternans: a report of 12 cases. Am J Med Sci. 1964 ; 248: 212-20.
15. OlopadeOI,UltmannJE. Malignanteffusions.CACancerJClin1991;41: 166-79.
16. Lalkhen AG, McCluskey A. Clinical tests: sensitivity and specificity. Continuing Educationin Anaesthesia, Critical Care&Crit Care & Pain J 2008; 8(6): 221-36.
17. Loong TW. Understanding sensitivity and specificity with the right side of the brain. BMJ 2003; 327: 716-19.
18. Spodick DH. Risk prediction inpericarditis: whotokeep in hospital? Heart 2008; 94(4): 398-9.