Zagazig University Medical Journal
www.zumj.journals.ekb.eg
Volume 28, Issue 6, November 2022(81-88) Supplement Issue

Manuscript ID ZUMJ-1907-1310 (R1)

DOI 10.21608/zumj.2019.14409.1310

ORIGINAL ARTICLE

Electrocardiographic Changes and Short-Term Prognosis in Patients with Acute Left Circumflex Artery Occlusion versus Right Coronary Artery Occlusion

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Submit Date 2019-10-28

Revise Date 2019-11-26

Accept Date 2019-12-09

ABSTRACT

Background: Acute myocardial infarction (AMI) is often caused by an atherosclerotic plaque rupture in one of the major epicardial coronary arteries. The difference in prognostic significance between anterior and inferior wall myocardial infarction has been thoroughly studied before.

Objective: To determine the clinical, angiographic, and prognostic characteristics associated with the different patterns of ST-segment changes in patients with left circumflex (LCX) artery occlusion versus right coronary artery (RCA) occlusion.

Methods: This prospective cross-sectional study included 78 patients with a first presentation of acute ST-elevation myocardial infarction (STEMI) and non-STEMI (NSTEMI), who were admitted to the Coronary Care Unit (CCU) of Cardiology Department at Zagazig University hospitals, Egypt, in the period from January 2018 to February 2019. Patients were divided into 2 main groups; Group 1: 41 patients with culprit lesion in LCX artery and Group2: 37 patients with culprit lesion in RCA artery.

Results: There was a statistically significant difference between studied groups regarding ejection fraction (EF), creatine phosphokinase (CK-MB) and troponin level (ST elevation significantly higher regard EF, CK-MB, and troponin level than other groups).

Conclusions: In patients with AMI due to culprits either in the LCX or RCA, ST elevation subgroup might be associated with 30-days major adverse cardiovascular events (MACE). Other independent predictors of 30-days MACE in such cohort include peak troponin level and LVEF. STEMI due to either a culprit in LCX or RCA may have similar 30-days MACE.

Keywords: Electrocardiography (ECG); Acute left circumflex artery (LCX) occlusion; Right coronary artery (RCA) occlusion; ST-elevation myocardial infarction (STEMI); Non-STEMI (NSTEMI).

INTRODUCTION

Acute myocardial infarction (AMI) is often caused by an atherosclerotic plaque rupture in one of the major epicardial coronary arteries. The difference in prognostic significance between anterior and inferior wall myocardial infarction has been thoroughly studied before [1, 2]. When the culprit coronary artery lesion is the left anterior descending artery (LAD), the infarction territory area and muscle damage is commonly larger than in either right coronary artery (RCA) or left circumflex (LCX) artery culprit lesion. This is because LAD perfuses larger myocardial territory area compared to either arteries, RCA or LCX [3]. Limited information exists about similar studies focusing on comparing myocardial infarction caused by RCA and LCX occlusion [4].Because of wide-spread use and low cost, 12-lead electrocardiogram (ECG) is one of the most useful tools in early diagnosis, risk stratifying and predicting in-hospital and long-term outcomes in different patterns of acute myocardial infarction [3].In many cases, ECG changes in the context of chest pain and acute coronary syndrome (ACS) may not be gross enough to guide treatment strategy leading to delay in offering primary intervention waiting for early elevation of cardiac biomarkers. This could have serious implications on prognosis and every effort should be exhausted to ensure early and timely intervention in such patients [5].Myocardial infarction (MI) can be divided into inferior, anterior, posterior, septal, and lateral. In addition, according to the ST elevation on the electrocardiogram (ECG), the myocardial

infarctions (MIs) are commonly categorized into ST elevation MI (STEMI) and non-ST elevation MI (NSTEMI) [4].Inferior wall MI is usually the result of either LCX or RCA artery occlusion [6]. The infarct-related artery (IRA) can significantly influence the disease progression of AMI patients [7].When RCA is shown to be the IRA, possible adverse effects include right ventricular infarct, complete heart block and other severe hemodynamic complications leading to shock, arrhythmias and even death [8].The three different ST-segment patterns observed in the admission ECG (ST- segment elevation, isolated depression, and no change) could distinguish patients at high and low risk of extensive infarction, heart failure, malignant ventricular arrhythmias, and other Major Adverse Cardiac Events (MACE) [9].

METHODS

This prospective cross-sectional study included 78 patients with a first presentation of acute STEMI and NSTEM, who were admitted to the Coronary Care Unit (CCU) of cardiology department at Zagazig University hospitals, Egypt, in the period from January 2018 to February 2019.

Ethical Approvals: Written informed consent was obtained from all participants and the study was approved by the Research Ethical Committee of Faculty of Medicine, Zagazig University, Egypt. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans. **Cases:** Patients were divided into 2 main groups; Group 1: 41 patients with culprit lesion in LCX artery and Group 2: 37 patients with culprit lesion in RCA artery. According to ST deviation on the admission ECG, each group was further subdivided into 3 subgroups: Group 1; Group 1a (21 patients): LCX was the culprit with ST elevation on ECG. Group 1b (11 patients): LCX was the culprit with ST depression > 0.1 mV in at least 2 consecutive leads. Group 1c (9 patients): LCX was the culprit with absence of ST shift, or ST shift <0.1 mV on ECG. Group 2; Group 2a (18 patients): RCA was the culprit with ST elevation on ECG. Group 2b (9 patients): RCA was the culprit with ST depression >0.1 mV in at least 2 consecutive leads. Group 2c (10 patients): RCA was the culprit with absence of ST shift, or ST shift <0.1 mV on ECG.

Inclusion Criteria: Patients with acute myocardial infarction (AMI) secondary to acute LCX or RCA occlusion, admitted within 12 hours of symptom onset.**Exclusion Criteria:**

Previous myocardial infarction or coronary artery bypass graft surgery, ventricular paced rhythm, bundle branch block, lack of available admission ECG or coronary angiography, more than mild valvular heart disease, significant systemic disease as renal failure, or liver call failure. Myocardial infarction was defined according to current fourth universal definition. STEMI was defined as a continuous chest pain that lasted >30 minutes, with the following criteria: ST segment elevation (0.2 mV) in right precordial leads or (>0.1 mV) in >2 contiguous leads on the 12-leads ECG, and elevated cardiac biomarker (Troponin T/I). The NSTMI was defined as ischemic symptoms in the absence of ST-segment elevation on the ECG with elevated cardiac markers [10].All patients were subjected to the following: **Complete history taking** including age, sex, history of coronary artery disease, risk factors including (hypertension, diabetes mellitus, smoking and family history of ischemic heart disease), chest pain, previous myocardial infarction, and previous stroke. **General and local examination** including pulse rate, rhythm, blood pressure (systolic & diastolic), and heart rate. Chest was examined for basal rales and Killip class. **Laboratory tests** including cardiac enzymes and Troponin: Blood samples for (Troponin-T, Creatine phosphokinase (CK) total, CK-MB, lactate dehydrogenase (LDH). Peak Troponin and CK-MB sample was obtained in the 1st 24-hours to assess the infarction size. Cardiac Troponin T was measured at presentation and 6 hours after symptom onset. Other laboratory tests including fasting plasma glucose, renal function tests, complete blood count (CBC). **A 12-Lead ECG:** A standard 12-lead electrocardiograms recorded at a speed of 25mm/s and caliber of 10mm/mV and ST shifts (at the J point), arrythmias, and heart rate were recorded. **Echocardiography:** During hospital admission, a 2-dimensional (2D) Echocardiography and Doppler was done to assess left ventricular (LV dimensions), mitral regurgitation (MR), ejection fraction (EF), and wall motion abnormalities (WMA). Machine used was Vivid E9 Ultrasound Machine with a multifrequency 1.7-4 MH2 transducer. All examinations were performed in the standard left lateral position. **Coronary angiography:** All patient had an invasive coronary angiography either during the primary percutaneous coronary intervention (PCI) (in ST elevation groups) or during the revascularization procedure in other groups afterwards. Standard Judkins catheters and angiographic projections were used. **For primary PCI:** Before the procedure, oral 600 mg clopidogrel was given to all patients in the cardiac catheterization room. Procedures were performed using standard angioplasty techniques with guiding catheters via the femoral artery. A bolus of 100 IU/kg heparin was administered after the insertion of the vascular access. Target lesions were initially treated with appropriate balloon predilatation if necessary,

followed by intracoronary stenting. **Follow-up:** Continuous follow-up of the patient was done within a period of 30 days after perfusion and catheterization. During hospital stay and after discharge, treatment plan and medication prescription were left to the treating physician and resident doctors. Significant variables that were associated with MACE included ST elevation on ECG, smoking, LVEF, triglycerides (TG), cholesterol and troponin levels. Independent predictors of MACE occurrence were ST elevation on ECG, Troponin level and LVEF.

Statistical Analysis: Investigative report form was used to register all demographic and clinical data, and these data were analyzed using Statistical Program for Social Science (SPSS) Version 12. Continuous data were tested by mean±SD, and categorial data by the count and percentage. Chi-Square test was used to detect relationship between 2 categorial variables. ANOVA (One Way Analysis of Variance) test to detect significant differences between the means of two or more independent groups. Level of significance: Significant when ($p<0.05$), highly significant when ($P<0.001$).

RESULTS

There was no statistically significant difference between the studied groups regarding age and heart rate (HR), while there was a statistically significant difference regarding systolic blood pressure (SBP) and diastolic blood pressure (DBP) (Table 1). There was no statistically significant difference between the studied groups regarding gender (Table 2). There was a statistically significant difference between the studied groups regarding previous stroke ($p=0.044$) (Table 3). There was a statistically significant difference between the studied groups regarding brady-arrhythmia, heart failure (HF) and MACE ($p=0.03$, $p=0.01$, 0.007 respectively) (Table 4). There was a statistically significant difference between the studied groups regarding left ventricular ejection fraction (LVEF) ($p=0.049$) (Table 5). There was a statistically significant difference between the studied groups regarding CK-MB and Troponin ($p=0.023$, $p=0.018$) (Table 6). There was no statistically significant difference between the studied groups regarding age, heart rate, SBP and DBP (Table S1). There was a statistically significant difference between the studied groups regarding heart failure and MACE ($p=0.03$, $p = 0.03$) (Table S2).

Table 1: The relation of RCA occlusion to the age and vital signs in the studied groups.

		N	Mean	Std. Deviation	F	P
Age (years)	ST elevation	18	57.83	10.70	0.073	0.930
	ST depression	9	58.33	7.29		
	Isoelectric ST	10	59.30	9.69		
Heart rate (beats/min)	ST elevation	18	78.55	15.87	1.234	0.304
	ST depression	9	70.77	8.08		
	Isoelectric ST	10	73.900	8.30		
Systolic blood pressure (mmHg)	ST elevation	18	137.77	21.77	4.289	0.022*
	ST depression	9	118.00	17.77		
	Isoelectric ST	10	123.10	8.18		
Diastolic blood pressure (mmHg)	ST elevation	18	87.50	11.91	7.167	0.003*
	ST depression	9	72.22	10.92		
	Isoelectric ST	10	77.50	6.41		

F (ANOVA) test. *p<0.05 Statistically significant.

Table 2: The relation of RCA occlusion to the gender in the studied groups.

Gender		ECG changes			Total	x^2	P
		Isoelectric ST	ST depression	ST elevation			
Female	N	3	3	3	9	1.14	0.56
	%	30.0%	33.3%	16.7%	24.3%		
Male	N	7	6	15	28		
	%	70.0%	66.7%	83.3.0%	75.7.0%		
Total	N	10	9	18	37		
	%	100%	100%	100%	100%		

x2 Chi-Squared. * p<0.05 Statistically significant.

Table 3: The relation of RCA occlusion to the risk factors in the studied groups.

		ECG changes			Total	x^2	P
		Isoelectric ST	ST depression	ST elevation			
Hypertension	N	4	4	10	18	0.7	0.702
	%	40.0%	44.4%	55.6%	48.6%		
Diabetes mellitus	N	8	6	10	24	1.702	0.42
	%	80.0%	66.7%	55.6%	64.9%		
Smoking	N	5	5	10	20	0.091	0.95
	%	50.0%	55.6%	55.6%	54.1%		
Previous stroke	N	4	0	2	6	6.25	0.044*
	%	40.0%	0.0%	11.1%	16.2%		
Total	N	10	9	18	37		
	%	100.0%	100.0%	100.0%	100.0%		

x2 Chi-Squared. * p<0.05 Statistically significant.

Table 4: The relation of RCA occlusion to the outcomes and complications in the studied groups.

			ECG changes			Total	X^2	P
			Iso-electric ST	ST depression	ST elevation			
Mitral regurgitation	Mild	N	2	4	12	18	6.45	0.206
		%	20.0%	44.4%	66.7%	48.6%		
	Moderate	N	1	0	0	1		
		%	10.0%	0.0%	0.0%	2.7%		
	Absent	N	7	5	6	18		
		%	70.0%	55.6%	33.4%	48.6%		
Brady-arrhythmia		N	1	1	9	11	6.4	0.03*
		%	10.0%	11.1%	50.0%	29.8%		
Atrial fibrillation		N	0	1	3	4	1.85	0.39
		%	0.0%	11.1%	16.7%	10.8%		
Ventricular Tachy-fibrillation		N	0	1	3	4	0.303	0.85
		%	0	11.1%	16.7%	13.5%		
Killip class	I	N	1	5	4	10	6.69	0.43
		%	10.0%	55.5%	22.2%	27.0%		
	II	N	7	3	8	18		
		%	70.0%	33.3%	44.4%	48.6%		
	III	N	2	1	6	9		
		%	20.0%	11.1%	33.3%	24.3%		
Heart failure		N	0	1	8	9	8.02	0.01*
		%	0.0%	11.1%	44.4%	24.3%		
MACE		N	0	1	9	10	9.61	0.007*
		%	0%	11.1%	50.0%	27.0%		
Mortality		N	0	0	3	3	2.11	0.265
		%	0.0%	0.0%	16.7%	8.1%		
Total		N	10	9	18	37		
		%	100.0%	100.0%	100.0%	100.0%		

x2 Chi-Squared. * p<0.05 Statistically significant. MACE (Major adverse cardiac events).

Table 5: The relation of RCA occlusion to the Echocardiographic parameters in the studied groups.

		N	Mean	Std. Deviation	F	P
Left ventricular end diastolic diameter (LVEDD) (mm)	ST elevation	18	51.1000	5.73298	1.304	0.285
	ST depression	9	51.8222	6.11428		

		N	Mean	Std. Deviation	F	P
	Isoelectric ST	10	47.9000	5.85852		
Left ventricular end systolic diameter (LVESD) (mm)	ST elevation	18	34.5389	4.97324	2.696	0.082
	ST depression	9	36.7444	5.23094		
	Isoelectric ST	10	30.8000	7.19259		
Left bentricular ejection fraction (LVEF) (%)	ST elevation	18	45.4000	5.67034	4.607	0.049*
	ST depression	9	49.8889	6.71648		
	Isoelectric ST	10	53.8722	4.64758		

Table 6: The relation of RCA occlusion to the laboratory parameters in the studied groups.

		N	Mean	Std. Deviation	F/ Kruskal Wallis	P
Cholesterol (mg/dl)	ST elevation	18	209.72	43.44	1.281	0.291
	ST depression	9	179.10	69.53		
	Isoelectric ST	10	210.40	41.32		
Triglycerides (mg/dl)	ST elevation	18	163.83	35.39	0.360	0.700
	ST depression	9	155.55	44.70		
	Isoelectric ST	10	171.70	48.37		
Blood urea (mg/dl)	ST elevation	18	47.77	41.07	0.917	0.409
	ST depression	9	29.44	9.32		
	Isoelectric ST	10	41.93	29.82		
Serum creatinine (mg/dl)	ST elevation	18	1.15	0.74	1.218	0.309
	ST depression	9	.82	0.26		
	Isoelectric ST	10	.89	0.42		
Fasting plasma glucose (mg/dl)	ST elevation	18	143.83	81.46	0.400	0.673
	ST depression	9	114.44	58.54		
	Isoelectric ST	10	139.50	99.50		
Serum Sodium Na (mmol/l)	ST elevation	18	137.05	4.68	0.163	0.851
	ST depression	9	137.00	5.29		
	Isoelectric ST	10	136.10	2.88		
Serum Potassium K (mmol/l)	ST elevation	18	4.20	0.50	0.150	0.861
	ST depression	9	4.14	0.49		
	Isoelectric ST	10	4.09	0.54		
Creatine phosphokinase CK-MB (ng/ml)	ST elevation	18	83.88	49.04	6.060	0.023*
	ST depression	9	37.83	17.13		
	Isoelectric ST	10	32.46	29.42		
Troponin (pg/ml)	ST elevation	18	419.39	86.70	7.774	0.018*
	ST depression	9	401.78	88.60		
	Isoelectric ST	10	359.50	130.93		

F (ANOVA) test/ KruskalWallis. *p<0.05 Statistically significant.

Table S1: The relation of LCX occlusion to the age and vital signs in the studied groups.

		N	Mean	Std. Deviation	F	P
Age (years)	ST elevation	21	53.85	7.94	0.867	0.428
	ST depression	11	56.09	5.57		
	Isoelectric ST	9	57.22	5.62		
Heart rate (beat/min)	ST elevation	21	80.71	14.34	0.020	0.980
	ST depression	11	79.81	12.09		
	Isoelectric ST	9	80.11	8.28		
Systolic blood pressure (mmHg)	ST elevation	21	140.47	20.18	1.752	0.187
	ST depression	11	130.72	20.53		
	Isoelectric ST	9	128.77	6.90		
Diastolic blood pressure (mmHg)	ST elevation	21	90.95	12.41	2.387	0.106
	ST depression	11	81.81	16.32		
	Isoelectric ST	9	83.00	6.55		

F (ANOVA) test. *p<0.05 Statistically significant.

Table S2: The relation of LCX occlusion to the outcome and complications in the studied groups.

			ECG changes			Total	X^2	P
			Isoelectric ST	ST depression	ST elevation			
Mitral regurgitation	Mild	N	3	4	14	21	9.54	0.059
		%	33.3%	36.4%	66.7%	51.2%		
	Moderate	N	0	0	3	3		
		%	0.0%	0.0%	14.3%	7.3%		
	Absent	N	6	7	4	17		
		%	66.7%	63.6%	19.0%	41.4%		
Brady- arrhythmia		N	0	0	1	1	0.97	0.61
		%	0.0%	0.0%	4.8%	2.4%		
Atrial fibrillation		N	0	0	3	3	3.2	0.11
		%	0.0%	0.0%	9.5%	7.3%		
Ventricular Tachy-fibrillation		N	0	1	2	3	0.91	0.63
		%	0.0%	9.1%	9.5%	7.3%		
Killip class	I	N	3	5	7	15	0.78	0.92
		%	33.3%	45.5%	33.3%	36.6%		
	II	N	4	4	7	14		
		%	44.4%	36.4%	33.3%	34.1%		
	III	N	2	3	7	12		
		%	22.3%	18.2%	33.3%	29.3%		
Heart failure		N	0	2	10	12	6.35	0.03*
		%	0.0%	18.2%	47.6%	29.3%		
MACE		N	0	2	10	12	6.35	0.03*
		%	0.0%	18.2%	47.6%	29.3%		
Mortality		N	0	0	3	3	3.2	0.11
		%	0.0%	0.0%	9.5%	7.3%		

x2 Chi-Squared. * p<0.05 Statistically significant. MACE (Major adverse cardiac events).

DISCUSSION

Acute myocardial infarction continues to be the major cause of morbidity and mortality worldwide. Despite major advances in diagnostic and prognostic tools over the past few decades, ECG remains the cornerstone in diagnosis and prognosis of myocardial infarction [11].The difference in prognostic significance between anterior and inferior wall myocardial infarction has been thoroughly studied before [1, 2]. The current study involved 78 patients who were diagnosed with acute MI due to LCX or RCA artery occlusion (41 patients in LCX group, 37 in RCA group). According to ST shifts, every group was further subdivided into ST elevation, ST depression and Isoelectric ST. We opted to enroll comparable number of patients in each LCX and RCA groups, so our patients were not consecutive. It is known that LCX-related STEMI only accounts for about 10% of all STEMI [12]. On comparing the 3 ST changes subgroups in the LCX group, we found no significant difference in demographic criteria or

coronary artery disease risk factors. This may be because the 3 subgroups share a common pathophysiologic chronic mechanism of atherosclerotic plaque formation and progression. This agrees with Vives-Borras et al. [12], who enrolled 314 patients with acute LCX artery occlusion and categorized them according to three ST segment patterns in the admission ECG. As expected, we found that the transmural ischemia associated with STEMI had an impact on both laboratory and echocardiographic parameters and subsequently on MACE compared to NSTEMI patients. In the LCX group, ST elevation subgroup was associated with significantly higher troponin and CK-MB level compared to either subgroups of ST depression or isoelectric ST segment. On echocardiography, this was reflected on a significant reduction regarding the EF in the STEMI group (41.3±5.08%). The correlation between troponin and CKMB quantitative value and the LV systolic dysfunction was detected [12]. Our results could have been more accurate if we used the Simpsons Rule method, and our finding of no difference in 30-day MACE in the ST elevation subgroups disagrees with Chin et al. [13], who studied 646 patients admitted with acute inferior wall STEMI and allocated to two groups according to culprit lesion either the LCX or the RCA. They found that the frequency of congestive heart failure, respiratory failure requiring mechanical support and the 30-day mortality were remarkably higher in LCX group. Chin et al. [13] also recommended using LCX occlusion as a risk-stratifying parameter in acute inferior STEMI patients undergoing primary PCI. They stated that the reason for the relatively poor prognosis outcome of LCX-related STEMI, compared RCA-related STEMI remains unclear. They partially attributed that to better developed collateral circulation and relatively better perfusion in the RCA group.Smith et al. [14] also studied 150 patients with acute inferior STEMI related to either LCX or RCA occlusion. They found that LCX patients had higher peak CK-MB levels, higher peak troponin, lower LVEF and more percentage of significant mitral regurgitation.On the other hand, Yip et al. [15] found that patients with inferior wall MI caused by dominant LCX occlusion had an unfavorable prognosis. Patients with LCX-related STEMI often have confusing ECG changes that could be of borderline significance. This could delay taking the decision of timely primary PCI and could have implication on prognosis afterwards. The discrepancy between the previous studies and our study could be attributed to small sample size, ethnic differences in our case. This could also be explained as we did not find a significant difference regarding right coronary circulation dominance in both groups.

On the other hand, we found that in both subgroups of ST elevation and ST depression, brady-arrhythmias were more common in the RCA-related MI, as agreed by Chin et al. [13] and Smith et al. [14]. This higher prevalence of brady-arrhythmias could be related to the fact that RCA supplies nodal branches and that a proximal culprit could cause ischemia in the node territory, subsequent brady-arrhythmias, and even serious advanced heart blocks. In the isoelectric ST subgroups, there was a benign course of cases in both groups. Risk-stratifying scores in NSTEMI patients as Thrombolysis in Myocardial Infarction (TIMI) and Global Registry of Acute Coronary Events (GRACE) give a better outcome to those with minor ECG changes [14]. However, in the isoelectric ST subgroup LCX-related MI patients had significant lower EF, but that was not translated into clinical events due to the normal mean of LVEF in both groups Using univariate and multivariate regression analysis, we found that the independent predictors of MACE in MI related to RCA or LCX arteries occlusion included ST elevation on ECG, higher troponin levels and lower LVEF. This agrees with Chin et al. [13], who found that on multivariate analysis, advanced CHF, high serum creatinine, lower LVEF, were independent predictors of 30-day MACE.Our results also agree with Sohrabi et al. [16], who found that troponin level, occurrence of MR, and lower LVEF were the independent predictors leading to poor outcome. Occurrence of acute MR could have its sudden loading effects on the previously unconditioned LV loading to acute LV failure and the need for mechanical and circulatory support.

CONCLUSIONS

In patients with AMI due to culprits either in the LCX or RCA, ST elevation cases might be associated with 30-days MACE. Other independent predictors of 30-days MACE in such cohort include peak troponin level and LVEF. STEMI due to either a culprit in LCX or RCA may have similar 30-days MACE.

Conflicts of Interest: Nothing to declare.

Financial Disclosures: Nothing to declare.

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To Cite:

Abdel-Samei, M., El-Zayat, A., Tawfik, O., Aljafaeri, T., Electrocardiographic Changes and Short-Term Prognosis in Patients with Acute Left Circumflex Artery Occlusion versus Right Coronary Artery Occlusion. Zagazig *University Medical Journal*, 2022; (81-88): -.doi: 10.21608/zumj.2019.14409.1310