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BRIEF ARTICLE

Prolonged QT dispersion in inflammatory bowel disease

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Abstract

AIM: To investigate the frequency and factors of prolonged QT dispersion that may lead to severe ventricular arrhythmias in patients with inflammatory bowel disease (IBD).

METHODS: This study included 63 ulcerative colitis (UC) and 41 Crohn's disease (CD) patients. Forty-seven healthy patients were included as the control group. Heart rate was calculated using electrocardiography, corrected QT dispersion (QTcd) and the Bazett's formula. Homeostasis model assessment (HOMA) was used to determine insulin resistance (IR). HOMA values < 1 were considered normal and values > 2.5 indicated a high probability of IR.

RESULTS: Prolonged QTcd was found in 12.2% of UC patients, and in 14.5% of CD patients compared with the control group ($P < 0.05$). A significant difference was found between the insulin values (CD: 10.95 ± 6.10 *vs* 6.44 ± 3.28, $P < 0.05$; UC: 10.88 ± 7.19 *vs* 7.20 ± 4.54, $P < 0.05$) and HOMA (CD: 2.56 ± 1.43 *vs* 1.42 ± 0.75, $P < 0.05$; UC: 2.94 ± 1.88 *vs* 1.90 ± 1.09, $P < 0.05$) in UC and CD patients with and without prolonged QTcd. Disease behavior types were determined in CD patients with prolonged QTcd. Increased systolic arterial pressure (125 ± 13.81 *vs* 114.09 ± 8.73, $P < 0.01$) and age (48.67 ± 13.93 *vs* 39.57 ± 11.58, $P < 0.05$) in UC patients were significantly associated with prolonged QTcd.

CONCLUSION: Our data show that IBD patients have prolonged QTcd in relation to controls. The routine follow-up of IBD patients should include determination of HOMA, insulin values and electrocardiogram examination.

Key words: Crohn's disease; Homeostasis model assessment; Insulin; QT dispersion; Ulcerative colitis

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INTRODUCTION

The most common extra-intestinal manifestations of Crohn's disease (CD) and ulcerative colitis (UC) are iridis, uveitis, primary sclerosing cholangitis, nodal erythema and pyoderma gangrenosum. Complications within the cardiovascular system seem to be uncommon, but there are no systematic investigations concerning the epidemiology of these manifestations. There have been more than 100 cases of pericarditis and perimyocarditis reported in patients with inflammatory bowel disease (IBD). Other patients with CD or UC suffer from vasculitis, representing a further mechanism of inflammatory diseases of the cardiovascular system. Some patients developed thrombotic complications due to activation of the coagulation system, which can result in atrial thrombi, embolism of the pulmonary arteries, myocardial infarction and disseminated intravascular coagulopa-

thy. Furthermore, a few cases of atrioventricular block, amyloidosis of the heart, dilated cardiomyopathy and endomyocardial fibrosis have been reported in patients with chronic IBD[1].

The most important causes of sudden death of cardiac origin are ventricular tachycardia and ventricular fibrillation cardiac arrhythmias. The action potentials in patients with long QT electrocardiographic (ECG) changes are not homogeneous, and as they result in the development of early and late repolarization facilitate the development of ventricular fibrillation[2]. The heterogeneity of QT dispersion (QTd) in ventricular repolarization is defined by the difference between the longest and shortest QT[3]. Increased QTd in many patients, and disease group were associated with the risk of serious arrhythmia and sudden death[2,4,5]. In patients with acute myocardial infarction, measurement of QT dispersion has shown little promise as a predictor of long-term mortality risk in individuals[6]. Cardiac involvement in IBD can cause serious ventricular arrhythmias with prolonged QTd, however, very little data is available on this subject[7]. In this study, QTd was prolonged in patients with IBD, and the relationships between disease characteristics and biochemical parameters were investigated.

MATERIALS AND METHODS

Ethics aspects

This study was formally authorized by the local Ethics Committee (approval date and number: 02-06-2009, No: 57). All of the patients gave written informed consent to the study protocol.

Study design

This was an observational, case-control study. Clinical, endoscopic, histologic, and radiologic findings showed that 63 patients had UC and 41 had CD. Forty-seven healthy control subjects were also included.

Selection of controls and patients

The control group had no chronic diseases, electrolyte imbalance and did not take regular medication. In addition, the controls were selected from persons with normal QT dispersion. IBD patients with known chronic disease, electrolyte imbalance, thyroid dysfunction or treated with drugs known to affect ECG findings were excluded from the study. Smoking status, alcohol habit, family history, use of medications, and physical examination were performed in all patients. After measurement of liver enzymes, blood count and blood levels of potassium, calcium and magnesium, those with abnormal findings were excluded from the study. To evaluate the clinical activity of CD, the Crohn's disease activity index (CDAI) was used. CDAI scores less than 150 indicated quiescence, while higher scores indicated active disease[8]. In UC patients, the Truelove-Witts clinical activity index was used to evaluate disease activity[9].

Anthropometric measurements

Anthropometric measurements (height, weight, waist circumference) were carried out by same person using standard instruments in patients wearing hospital clothing, while standing. Body mass index (BMI) was calculated by dividing the patient's weight by the square of his or her height (mass/height^2, kg/m^2). Waist circumference was measured while standing at the narrowest area of the waist midway between the anterior superior iliac spine and the lower rib margin on light expiration. Average systolic and diastolic blood pressure measurements were recorded. Systolic blood pressure (SBP) of 140 mmHg and over and diastolic blood pressure of 90 mmHg and over were accepted as hypertension[10]. Patients with hypertension were excluded.

QT interval calculation

All patients and controls underwent 12-lead surface ECG to determine QT interval, and the QRS complex was defined as the distance from the beginning to the end of the T wave. Heart rate, physiological causes of shortening of the QT interval, and reduction in speed will cause QT prolongation. Various formulations of the QT interval were corrected for heart rate. Corrected QT (QTc) was calculated according to the Bazett's formula (Bazett's formula QTc = QT × $\sqrt{RR}$). QTd and minimum mean QT as the difference between the average QT were also calculated. Corrected QT dispersion (QTcd) values were calculated in the same way. Extended QTc interval was defined as a duration of > 440 ms[11].

Biochemical analysis

After 12 h of night fasting, venous blood samples were taken, centrifuged (2500 cycle/min) and the serum separated to measure biochemical indices. Glucose, total cholesterol, low density lipoprotein cholesterol, high density lipoprotein cholesterol, triglyceride and uric acid levels were determined by enzymatic methods, and insulin level was determined by electrochemiluminescence immunoassay using a Roche E170 device. Insulin sensitivity was assessed by homeostasis model assessment of insulin resistance (HOMA-IR). Accordingly, IR was calculated using the following formula: Fasting insulin level (μIU/mL) × fasting plasma glucose (mmol/L)/22.5[12].

Statistical analysis

All data are presented as mean ± SE. Data were analyzed using descriptive statistical methods (mean, standard deviation, frequency). Differences between CD, UC, and controls, demographic, anthropometric, and laboratory values and the relationship between QT duration were calculated using the Kruskal-Wallis test, Mann-Whitney *U* test, and Chi-square test. Pearson correlation analysis was used to determine the relationship between variables. All analysis were performed using SPSS version 15.0 (SPSS Inc., United States). Statistical significance was accepted at $P \leq 0.05$.

Table 1 Characteristics of patients with Crohn's disease, and the relationship between QT prolongation *n* (%)

	CD QT prolongation		Test values	
	-	+	χ^2	*P* value
Diagnosis period (yr)	26.47 ± 34.27	27.2 ± 32.88	*Z* = 0.31	0.74
Family history			0.97	0.61
None	30 (83.3)	5 (100)		
CD	3 (8.3)			
Colon cancer	3 (8.3)			
Operation			2.11	0.14
Yes	5 (13.88)	2 (40)		
No	31 (86.11)	3 (60)		
Age at diagnosis (yr)			3.01	0.22
< 17	2 (5.6)			
17-40	33 (91.70)	4 (80)		
> 40	1 (2.80)	1 (20)		
Disease behavior			23.35	0.000[b]
Non-stricturing	22 (61.1)	1 (20)		
Non penetrating				
Stricturing	14 (38.9)	1 (20)		
Penetrating	-	3 (60)		
Disease location			0.55	0.75
İleal	12 (33.3)	1 (20)		
Colonic	1 (2.80)	-		
İleocolonic	23 (63.90)	4 (80)		
CDAI value			0.013	0.91
CDAI < 150	28 (77.7)	4 (80)		
CDAI ⩾ 150	8 (22.2)	1 (20)		
Drug use			0.13	0.71
Used	26 (72.2)	4 (80)		
Not used	10 (27.8)	1 (20)		
5-ASA			0.23	0.62
Used	11 (30.6)	1 (20)		
Not used	25 (69.4)	4 (80)		
CS			0.97	0.32
Used	30 (83.3)	5 (100)		
Not used	6 (16.7)	-		
IS			0.13	0.71
Used	26 (72.2)	4 (80)		
Not used	10 (27.8)	1 (20)		
Smoking			0.66	0.72
Yes	21 (58.3)	2 (40)		
No	9 (25)	2 (40)		
Quit	6 (16.7)	1 (20)		
Alcohol			2.08	0.35
Yes	9 (25)	-		
No	25 (69.4)	5 (100)		
Quit	2 (5.6)	-		

[b]$P < 0.01$, χ^2 test and *Z*: Mann-Whitney *U* test. CD: Crohn's disease; CDAI: Crohn's disease activity index; 5-ASA: 5-aminosalicylic acid; CS: Corticosteroid; IS: immunosuppressive.

Table 2 Characteristics of patients with ulcerative colitis, and the relationship between QT prolongation *n* (%)

	UC QT prolongation		Test values	
	-	+	χ^2	*P* value
Diagnosis period (yr)	44.13 ± 42.60	45.67 ± 47.99		0.86
Family history			0.48	0.78
None	41 (77.4)	6 (66.7)		
UC	8 (15.1)	2 (22.2)		
Colon cancer	4 (7.5)	1 (11.1)		
Operation			0.17	0.67
Yes	1 (1.9)	-		
No	52 (98.1)	9 (100)		
Extent of disease			3.49	0.32
Rectosigmoid	16 (30.18)	2 (22.22)		
Left sided colon	10 (18.86)	4 (44.4)		
Extensive	6 (11.32)	-		
Pancolitis	21 (39.62)	3 (33.3)		
Clinical activity			2.19	0.33
TW1 (mild activity)	34 (64.15)	8 (88.89)		
TW2 (moderate activity)	17 (32.07)	1 (11.11)		
TW3 (severe activity)	3.77	-		
Drug use			0.09	0.75
Used	45 (84.9)	8 (88.89)		
Not used	8 (15.09)	1 (11.11)		
5-ASA			0.19	0.65
Used	9 (16.98)	1 (11.11)		
Not used	44 (83.01)	8 (88.89)		
CS			1.12	0.28
Used	47 (88.67)	9 (100)		
Not used	6 (11.32)	-		
IS			0.13	0.71
Used	49 (92.45)	8 (88.89)		
Not used	4 (7.54)	1 (11.11)		
Smoking			2.99	0.22
Yes	12 (22.64)	2 (22.2)		
No	26 (49.05)	2 (22.2)		
Quit	15 (28.30)	5 (55.56)		
Alcohol				
Yes	9 (16.98)	2 (22.2)	0.46	0.79
No	42 (79.24)	7 (77.78)		
Quit	2 (3.77)	-		

χ^2 test, Mann-Whitney *U* test. TW: Truelove-Witts clinical activity index; UC: Ulcerative colitis; 5-ASA: 5-aminosalicylic acid; CS: Corticosteroid; IS: Immunosuppressive.

RESULTS

Demographic characteristics

This study included 63 UC patients (33 female, 30 male; mean age 40.89 ± 12.25 years), 41 CD patients (16 female, 25 male; mean age 38 ± 7.79 years) and 47 healthy controls (25 female, 32 male; mean age 41.21 ± 10.55 years).

Descriptive analysis of patients

Of these patients; 12.2% UC and 14.5% CD patients had prolonged QTcd when compared with the control group ($P < 0.05$). Increased SBP ($P < 0.01$) and age ($P < 0.05$) in UC patients were significantly associated with prolonged QTcd. Disease group, disease duration, family history, smoking, alcohol use, history of surgery, disease activity, localization, and drug use were not related to prolonged QTcd ($P > 0.05$). Disease behavior type was determined to be involved in CD patients with prolonged QTcd ($P < 0.001$) (Tables 1 and 2).

Biochemical analysis and the relationship between prolonged QT intervals

Significant differences were observed in patients with UC and CD with respect to insulin values ($P < 0.05$) and HOMA ($P < 0.05$) values compared to patients without UC and CD (Tables 3 and 4). Both groups demonstrated HOMA values above 2.5 (Figure 1). Using Pearson cor-

Table 3 Crohn's disease and control group, demographic, anthropometric and laboratory values and the relationship between QT prolongation

	CD QT prolongation (-)	CD QT prolongation (+)	Control	Test values
Male, *n* (%)	23 (63.9)	2 (40)	31 (66)	$\chi^2 = 1.31$, $P = 0.51$
Female, *n* (%)	13 (36.1)	3 (60)	16 (34)	
Age (yr)	36.80 ± 7.79	41.33 ± 7.66	41.21 ± 10.55	$P = 0.53$
SBP (mmHg)	109.72 ± 13.41	105 ± 20	102.74 ± 11.93	$P = 0.071$
DBP (mmHg)	69.72 ± 9.40	70 ± 10	71.11 ± 6.55	$P = 0.62$
Heart rate (beats/min)	77.13 ± 13.4	71.4 ± 12.23	74.80 ± 13.25	$P = 0.36$
BMI (kg/m²)	23.78 ± 4.90	25.38 ± 3.99	24.77 ± 3.43	$P = 0.57$
WC (cm) male	89 ± 7.88	91 ± 6.36	90.19 ± 4.99	$P = 0.14$
WC (cm) female	87.23 ± 5.45	92.33 ± 7.08	90.68 ± 5.18	$P = 0.19$
QTcd (ms)	39.69 ± 2.32	55.4 ± 2.88	38.68 ± 2.25	CD QT prolongation (-) *vs* (+), CD QT prolongation (+) *vs* control, $^bP = 0.000$; CD QT prolongation (-) *vs* control, $^aP = 0.049$
FPG (mg/dL)	89.31 ± 6.87	93.8 ± 10.75	87.6 ± 10.13	$P = 0.078$
Total-C (mg/dL)	187.89 ± 39.46	189.8 ± 21.28	185.68 ± 37.74	$P = 0.65$
Triglyceride (mg/dL)	116.51 ± 64.85	111 ± 33.12	97.98 ± 36.74	$P = 0.50$
HDL-C (mg/dL)	55.4 ± 12.69	60 ± 19.19	58.17 ± 14.74	$P = 0.66$
LDL-C (mg/dL)	121.05 ± 32.66	118.2 ± 9.78	119.02 ± 24.48	$P = 0.77$
Insulin (μU/mL)	6.44 ± 3.28	10.95 ± 6.10	5.68 ± 3.83	CD QT prolongation (-) *vs* (+), $^aP = 0.036$; CD QT prolongation (-) *vs* control, $^aP = 0.027$; CD QT prolongation (+) *vs* control, $P = 0.16$
HOMA-IR	1.42 ± 0.75	2.56 ± 1.43	1.31 ± 0.94	CD QT prolongation (-) *vs* (+), $^aP = 0.026$; CD QT prolongation (-) *vs* control, $P = 0.12$; CD QT prolongation (+) *vs* control, $^aP = 0.023$
Uric acid (mg/dL)	4.09 ± 0.86	4.26 ± 1.52	4.01 ± 1.05	$P = 0.95$

$^aP < 0.05$, $^bP < 0.01$, χ^2 test, Mann-Whitney *U* test, Kruskall-Wallis test. CD: Crohn's disease; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; BMI: Body mass index; WC: Waist circumference; QTcd: Corrected QT dispersion; FPG: Fasting plasma glucose; C: Cholesterol; HDL: High density lipoprotein; LDL: Low density lipoprotein; HOMA-IR: Homeostasis model assessment of insulin resistance.

Table 4 Ulcerative colitis and control group, demographic, anthropometric, and laboratory values and the relationship between QT prolongation

	UC QT prolongation (-)	UC QT prolongation (+)	Control	Test values
Male, *n* (%)	25 (47.2)	5 (55.6)	31 (66)	$\chi^2 = 3.56$, $P = 0.16$
Female, *n* (%)	28 (52.8)	4 (44.4)	16 (34)	
Age (yr)	39.57 ± 11.58	48.67 ± 13.93	41.21 ± 10.55	$P = 0.14$
SBP (mmHg)	114.09 ± 8.73	125 ± 13.81	102.74 ± 9.93	UC QT prolongation (-) *vs* (+), $^aP = 0.012$; UC QT prolongation (-) *vs* control, $P = 0.51$; UC QT prolongation (+) *vs* control, $^aP = 0.037$
DBP (mmHg)	73.96 ± 6.88	74.44 ± 8.81	71.11 ± 6.55	$P = 0.66$
Heart rate (beats/min)	77.13 ± 13.4	71.4 ± 12.23	74.80 ± 13.25	$P = 0.25$
BMI (kg/m²)	25.22 ± 4.31	27.72 ± 9.11	24.77 ± 3.43	$P = 0.18$
WC (cm) male	94.92 ± 9.91	104.6 ± 22.67	93.19 ± 10.99	$P = 0.59$
WC (cm) female	92.75 ± 12.83	93.5 ± 10.96	90.68 ± 12.18	$P = 0.54$
QTcd (ms)	40.09 ± 2.34	55.1 ± 3.1	38.68 ± 2.25	UC QT prolongation (-) *vs* (+), UC QT prolongation (+) *vs* control, $^bP = 0.000$; UC QT prolongation (-) *vs* control, $^bP = 0.003$
FPG (mg/dL)	93.62 ± 9.02	95.11 ± 17.33	87.6 ± 10.13	$P = 0.53$
Total-C (mg/dL)	194.49 ± 34.31	214.33 ± 40.84	185.68 ± 37.74	$P = 0.34$
Triglyceride (mg/dL)	114.33 ± 53.89	120.11 ± 36.31	97.98 ± 36.74	$P = 0.10$
HDL-C (mg/dL)	54.62 ± 15.10	61.22 ± 14.95	58.17 ± 14.74	$P = 0.38$
LDL-C (mg/dL)	124.81 ± 24.41	137.552 ± 28.75	119.02 ± 24.48	$P = 0.49$
Insulin (μU/mL)	7.20 ± 4.54	10.88 ± 7.19	5.68 ± 3.83	UC QT prolongation (-) *vs* (+), $^aP = 0.031$; UC QT prolongation (-) *vs* control, $P = 0.43$; UC QT prolongation (+) *vs* control, $^aP = 0.047$
HOMA-IR	1.90 ± 1.09	2.94 ± 1.88	1.31 ± 0.94	UC QT prolongation (-) *vs* (+), $^aP = 0.011$; UC QT prolongation (-) *vs* control, $P = 0.51$; UC QT prolongation (+) *vs* control, $^aP = 0.027$
Uric acid (mg/dL)	4.31 ± 1.35	4.44 ± 1.29	4.01 ± 1.05	$P = 0.12$

$^aP < 0.05$, $^bP < 0.01$, χ^2 test, Mann-Whitney *U* test, Kruskall-Wallis test. UC: Ulcerative colitis; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; BMI: Body mass index; WC: Waist circumference; QTcd: Corrected QT dispersion; FPG: Fasting plasma glucose; C: Cholesterol; HDL: High density lipoprotein; LDL: Low density lipoprotein; HOMA-IR: Homeostasis model assessment of insulin resistance.

relation analysis, a moderately significant positive relationship was observed between QT time in patients with CD and UC and insulin (CD: $r = 0.542$, $P = 0.023$; UC: $r = 0.560$, $P = 0.021$) and HOMA values (CD: $r = 0.618$, $P = 0.018$; UC: $r = 0.678$, $P = 0.011$).

DISCUSSION

Homogeneous ventricular resting time is believed to be protective against arrhythmias[13]. Easy and non-invasive methods to determine the risk of ventricular arrhythmia,

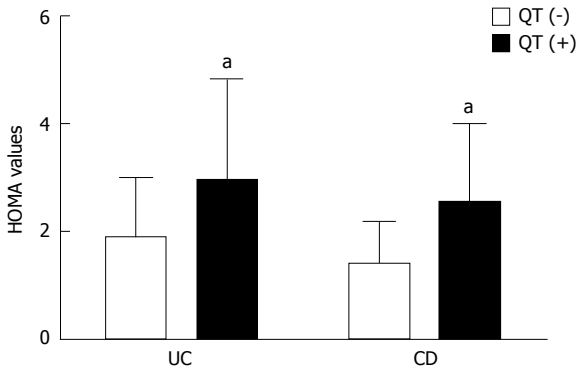

Figure 1 Homeostasis model assessment values in ulcerative colitis and Crohn's disease patient. Data are shown as mean ± SD. $^{a}P < 0.05$ *vs* QT prolongation (-) groups using Mann-Whitney *U* test. HOMA: Homeostasis model assessment; UC: Ulcerative colitis; CD: Crohn's disease.

are an advantage in patients with QTd. However, the frequency and prognostic value of IBD as an indicator of arrhythmia is not fully understood. In this study, the demographic data of patients with IBD and prolonged QT dispersion and the relationships with some cardiovascular risk parameters were investigated. Conflicting results were obtained in previous studies investigating the relationship between age and QT interval. Some studies in older healthy individuals showed a significant correlation between age and prolonged QTd[14-16]. In contrast, Merri *et al*[17] looked at descriptive ECG features of ventricular repolarization, and did not find a relationship between age and QT interval. The relationship between gender and QT interval has also been investigated. In the literature, some studies have reported long-QT dispersion in healthy women compared with men[18,19]. Mangoni and colleagues found no differences between QTd and gender[14]. In our study, UC patients showed a significant relationship between older age and prolonged QTd. Smoking may predispose to ventricular fibrillation and sudden cardiac death by altering ventricular recovery time dispersion indices. Some studies found a correlation between smoking in men and QT dispersion, this was not detected in the study by Mangoni and colleagues[14,20,21]. In our study, both UC and CD patients did not show a correlation between smoking and prolonged QTd. Drugs such as steroids and azathioprine can be cardiotoxic[22-27]. There was no correlation between QT dispersion and the use of 5-aminosalicylic acid, steroids and azathiopurine. In fact, although UC and CD are generally described as two expressions of a common pathophysiological process, they may be characterized by different autonomic alterations due to the diverse location of inflammatory lesions along the gastrointestinal tract[28]. Inflammation tends to be diffuse and uniform in UC, mainly affecting the superficial layers of the inner lining of the bowel, where autonomic neural fibers of the submucosal plexus are located[29], whereas in CD inflammation is concentrated in some areas more than in others, involving deeper layers of the bowel wall. The different location and structure of the lesions may therefore have different effects on autonomic gastrointestinal control, possibly reflected by differences in cardiovascular autonomic control[30]. In our study, prolonged QTd was significantly more frequent in patients with stricturing and penetrating CD. The relationship between physiologic BMI and QT interval confirms and extends the findings of previous reports which showed a positive relationship between these variables in human obesity[31-33]. Prolonged QTcd was observed in about 30% of subjects with impaired glucose tolerance in a report from the NHANES Ⅲ cohort, and a positive association was found between BMI and QTcd[34]. Some studies have also found a positive correlation between waist circumference and QTd[31,35,36]. In our study, prolonged QTd was found in UC and CD patients with higher BMI and waist circumference, although no statistically significant relationship was found. IR is a metabolic disorder characterized by a reduction in the use of glucose in skeletal muscle. Excess sodium retention, insulin-like cellular proliferation and matrix expansion causes vascular responses. Insulin, various growth factors, and vascular damage can accelerate atherosclerosis[37]. Nigro and colleagues found that HOMA and insulin concentrations were significantly correlated with QTd[33]. In our study, UC and CD patients with high insulin and HOMA values showed a statistically significant relationship with QT dispersion. The two groups demonstrated HOMA values above 2.5. According to JNC 7, subjects with systolic blood pressure of 120-139 mmHg are considered to have prehypertension[8]. Some studies showed a significant relationship between subjects with prehypertension and prolonged QTd[38,39]. In our study, SBP in UC patients with prolonged QTd was significantly higher than that in those without prolonged QTd, and the controls. QT dispersion seen before the development of hypertension and diabetes mellitus may be associated with IBD etiopathogenesis.

Simple, inexpensive and non-invasive methods can be used to identify QTd, ventricular arrhythmias, and the risk of cardiovascular diseases. These cardiovascular diseases may occur as a result of prolonged QTd, and IBD exacerbates ventricular arrhythmias and can cause sudden death. In IBD patients, prolonged QTd can be used to determine cardiovascular risk factors thought to be useful for the large patient groups in controlled trials.

COMMENTS

Background

Inflammatory bowel disease (IBD) involves two separate chronic idiopathic inflammatory diseases: Crohn's disease and ulcerative colitis. Increased QT dispersion (QTd) is a marker of myocardial electrical instability and predicts ventricular arrhythmias and sudden cardiac death.

Research frontiers

Easy and non-invasive methods to determine the risk of ventricular arrhythmia are an advantage in patients with QTd. However, the frequency and prognostic value of IBD as an indicator of arrhythmia is not fully understood. In this study, the demographic data of patients with IBD and prolonged QT dispersion and their relationship with some cardiovascular risk parameters were investigated.

Innovations and breakthroughs

Cardiac involvement in IBD can cause serious ventricular arrhythmias with prolonged QTd, however, very little data is available on this subject. QT disper-

sion is prolonged in patients with IBD. The relationship between QT dispersion, insulin, homeostasis model assessment (HOMA) and increased cardiovascular risk factors were investigated in this study.

Applications

Determination of the factors causing prolongation of the treatment of patients with IBD with QT dispersion and reorganization will provide additional benefit in the future prognosis of these diseases.

Peer review

The aim of the study was to investigate the whether prolonged QT dispersion as measured on the electrocardiographic, at risk of developing ventricular fibrillation, in adult patients with IBD. In addition, the authors determined the level of insulin resistance in these patients using HOMA.

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