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değildir. Tahmin edileceği ve birçok çalışmada gösterildiği üzere hastalarda ki değişim, bu üç girişimin ortak ya da kümülatif etkisi, bu müdahalelerin birinin oluşturacağı etkiden daha büyük olacaktır (3-5).

Koroner anjiyografinin durumsal anksiyeteyi arttıran bir durum olması, koroner arter hastalığı tanısı alan hastaları eğitilmeye hazır hale getirmesi, çalışmamızda göz ardı edilen bir durum değildir. Kaldı ki, orta düzeyli anksiyetenin eğitime hazırlayıcı, yüksek düzeydeki anksiyetenin ise eğitimin etkisini azaltacağı gösterilmiştir. Bu durum yalnızca koroner arter hastaları için değil, tüm kronik hastalara verilecek eğitim çalışmalarında etkili bir faktördür. Çalışmanın tasarımı gereği, kontrol grubu kullanılmaması, kontrol ve hasta grupları arasında var olabilecek potansiyel anksiyete düzey farklılığından ya da başka farklılıklardan doğacak hataları oluşturmaması da hastaların kendisiyle (müdahale öncesi ve sonrası) karşılaştırılmasının başka bir avantajıdır (6, 7).

Araştırmamızda hastalara eğitim verilirken, Prochaskanın 1977'de tanımladığı transteoretik model, davranış değişikliğinin hangi evresinde nasıl eğitim verileceği konusunda iyi bir rehber olan davranış değişikliğinin evresine değinilmemiştir. Ancak sağlık inanç modeli, planlı davranış modeli vb. davranış değişikliği modelleri de kullanılmamıştır. Bunun nedeni bu araştırmamızda tek bir davranışsal soruna yönelik değil, alkol, sigara, obezite, egzersiz sürelerinin arttırılması, sağlıklı vücut ağırlığının korunması gibi birden çok davranış değişikliği ile uğraşmanın istatistiksel yönden analiz edilmesi ve yorumlanması imkansız sayıda hasta grubu oluşturacağı dikkate alınması gerekir (7).

Sonuç olarak; araştırmamızın koroner arter hastalarına birden çok konuda tek bir sağlık personelinin verdiği kısa süreli eğitim ve danışmanlığının davranış değişikliği oluşturup oluşturmadığının ortaya koyulması amaçlanmış ve obezite, egzersiz sürelerinin arttırılması, sağlıklı vücut ağırlığının korunmasında ve bazı klinik verilerde düzelmelerin bu tür bir yaklaşımın yararlı sonuçlar oluşturulabileceği gösterilmiştir. Alkol ve sigara gibi farklı bağımlılık biçimleri için davranışlarda düzelmenin olmayışı da, bu tarz bir eğitimin yetersiz kalacağını gösteren diğer bulgulardır.

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Kaynaklar

1. Kurçer MA, Özbay A. Koroner arter hastalarında uygulanan yaşam tarzı eğitim ve danışmanlığının yaşam kalitesine etkisi. Anadolu Kardiyol Derg 2011; 11: 107-13.
2. Fletcher RH, Fletcher SW, Wagner EH. Clinical Epidemiology William and Wilkins, Maryland, 1996. p.158-61.
3. Boulay P, Prud'homme PD. Risk factor management after short-term versus long-term cardiac rehabilitation program. Coronary Health Care 2001; 5: 133-40.
4. Lai SC, Cohen MN. Promoting lifestyle changes. Am J Nurs1999; 4: 63-73.
5. Sheridan SL, Viera AJ, Krantz MJ, Ice CL, Steinman LE, Peters KE, et al. The effect of giving global coronary risk ınformation to adults. Arch Intern Med 2010; 170: 230-9.
6. Uzun S, Vural H, Uzun M, Yokuşoğlu M. State and trait anxiety levels before coronary angiography. J Clin Nurs 2008; 17: 602-7.
7. Glanz K, Lewis FM, Rimer BK, editors. Health Behavior and Health Education. 2nd ed. San Fransisco: Josey Bass Inc; 1997.

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Available Online Date/Çevrimiçi Yayın Tarihi: 05.07.2011

QT interval prolongation due to metronidazole administration

Metronidazol uygulamasına bağlı QT uzaması

The drug induced Q-T prolongation might lead to torsades de pointes (TdP), ventricular tachycardia and sudden cardiac death. Several antimicrobial agents, such as erythromycin, clarithromycin, fluoroquinolones, halofantrine and pentamidine can cause QT prolongation. Metronidazole is a widely used antimicrobial agent and the effect of this drug on QT interval is not well documented (1).

A 71-year-old female patient was admitted to emergency unit with severe dyspnea, angina, and orthopnea symptoms in the preceding 5 days. She was on treatment for chronic obstructive lung disease, diabetes mellitus, hypertension and coronary artery disease. She also had a history of drug-induced QT prolongation associated with moxifloxacin. Electrocardiogram (ECG) on admission showed atrial fibrillation with a ventricular rate of 82 /minute. The corrected Q-T interval was 396 msec (Fig. 1). She was treated with diuretics, angiotensin converting enzyme inhibitors, omeprazole and bronchodilators. On follow up diagnosis of nosocomial pneumonia was made and intravenous metronidazole was initiated. Two days after the initiation of antimicrobial therapy, the QT interval prolonged (corrected Q-T interval, 559 msec) and the T waves were inverted (Fig. 2). Laboratory tests, including serum potassium and magnesium levels, were within normal limits. Metronidazole was immediately stopped and the ECG of the patient returned to normal within 48- hours.

Commonly prescribed drugs, such as antibiotics, psychotropic agents and histamine H1-receptor antagonists, can prolong cardiac repolarization and might trigger a polymorphic ventricular tachycardia called torsades de pointes (1). Azole derivatives including ketoconazole, itraconazole and fluconazole can also prolong QT and trigger TdP directly or by interacting with other QT prolonging agents (2).

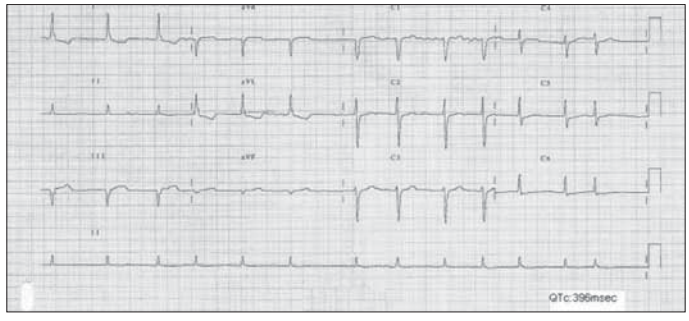

Figure 1. Electrocardiogram before metronidazole administration (Corrected Q-T interval: 396 msec)

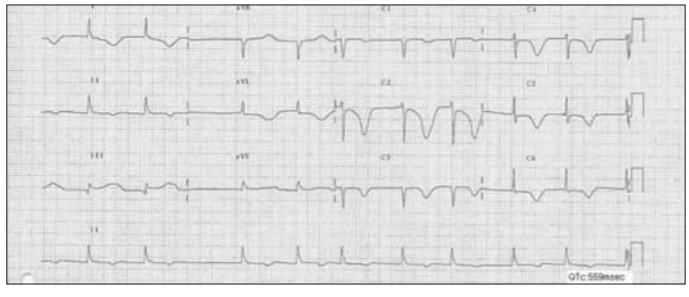

Figure 2. Electrocardiogram after metronidazole administration (Corrected Q-T interval: 559 msec)

The mechanism of QT prolongation during metronidazole administration is unclear. Metronidazole is a widely used antimicrobial medication and is a potent inhibitor of CYP3A4 and CYP2C9 isoenzymes. Theoretically, it may cause QT prolongation by inhibiting the metabolism of other drugs that have potential to cause QT prolongation. The patient was also taking omeprazole. Omeprazole is also a competitive inhibitor of the enzymes CYP2C19 and CYP2C9, and may therefore interact with drugs that depend on them for metabolism (3). As a result of this mechanism metronidazole with the concomitant use of omeprazole may cause indirectly QT prolongation and TdP through its interaction with other QT prolonging agents (4). However, in our case, the patient was not taking any other medications that may interact with these enzymes or have potential to cause QT prolongation. We thought that metronidazole caused QT prolongation by another mechanism that we do not know. There is only one case in the literature showing that metronidazole alone can cause QT prolongation (5).

In our patient there were no predisposing factors for QT prolongation such as electrolyte imbalance or administration of other medications known to prolong QT interval. There were no medications discontinued within a relatively short time line before initiating metronidazole. She had similar problems in the past associated with fluoroquinolone use and might therefore be a carrier of a silent mutation in one of the congenital long QT syndrome-associated genes.

These patients are at high risk for developing QT prolongation and TdP when exposed to drugs which affect potassium channels. As a result the arrhythmogenic properties of metronidazole should be evaluated carefully, and avoid prescribing to patients at high risk for drug-induced TdP, such as elderly patients with structural heart disease, renal failure or impaired liver function.

Metronidazole is widely used antimicrobial agent and the potential of this agent to prolong QT is not well documented. This case report shows that intravenous metronidazole can prolong QT interval in susceptible patients and it should be used with close monitoring of the ECG.

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References

1. Viskin S, Justo D, Halkin A, Zeltser D. Long QT syndrome caused by noncardiac drugs. Prog Cardiovasc Dis 2003; 45: 415-27.
2. Justo D, Zeltser D. Torsade de pointes induced by systemic antifungal agents: lessons from a retrospective analysis of published case reports. Mycoses 2006; 49: 463-70.
3. Stedman CA, Barclay ML. Review article: comparison of the pharmacokinetics, acid suppression and efficacy of proton pump inhibitors. Aliment Pharmacol Ther 2000; 14: 963-78.
4. Kounas SP, Letsas KP, Sideris A, Efraimidis M, Kardaras F. QT interval prolongation and torsades de pointes due to a coadministration of metronidazole and amiodarone. Pacing Clin Electrophysiol 2005; 28: 472-3.
5. Cohen O, Saar N, Swartzon M, Kliuk-Ben-Bassat O, Justo D. First report of metronidazole-induced QT interval prolongation. Int J Antimicrob Agents 2008; 31: 180-1.

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Available Online Date/Çevrimiçi Yayın Tarihi: 05.07.2011

doi:10.5152/akd.2011.120

Thirty-six years with the same prosthetic mitral valve

Aynı mitral protez kapakla otuz altı yıl

A 62-years-old female patient presented to our clinic with right heart failure. On her past medical history there was a history mitral valve surgery 36 years ago, which was performed for replacement of stenotic rheumatic mitral valve with Starr-Edwards cage-balled prosthetic valve. She was doing well except a history of transient ischemic attack nearly 30 years ago. Recently she developed clinical signs of right heart failure. On echocardiogram there was an excellent working prosthetic mitral valve, with gradients of 12/6 mmHg (maximum/mean) and no mitral regurgitation (Fig. 1). However, it was seen that she had severe tricuspid stenosis with gradients of 15/7 mmHg (maximum/mean) and also severe tricuspid regurgitation. After compensation of patient, coronary angiography and ventriculography was performed and patient was scheduled for tricuspid valve surgery (Fig. 2, 3).

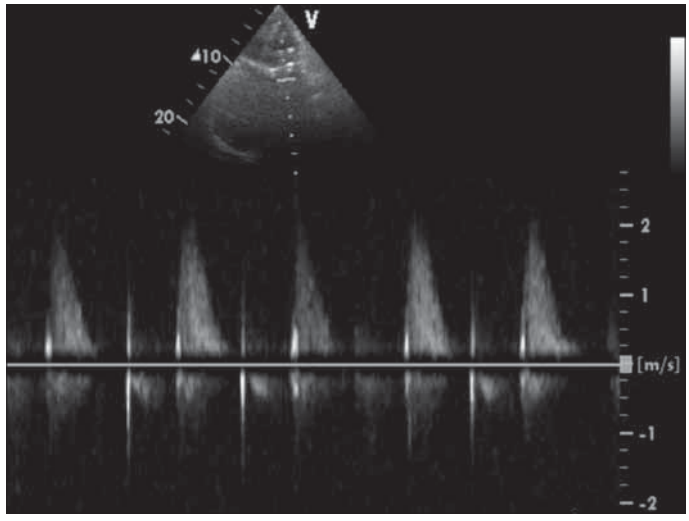

Figure 1. Transthoracic Doppler echocardiographic view of mitral valve gradients

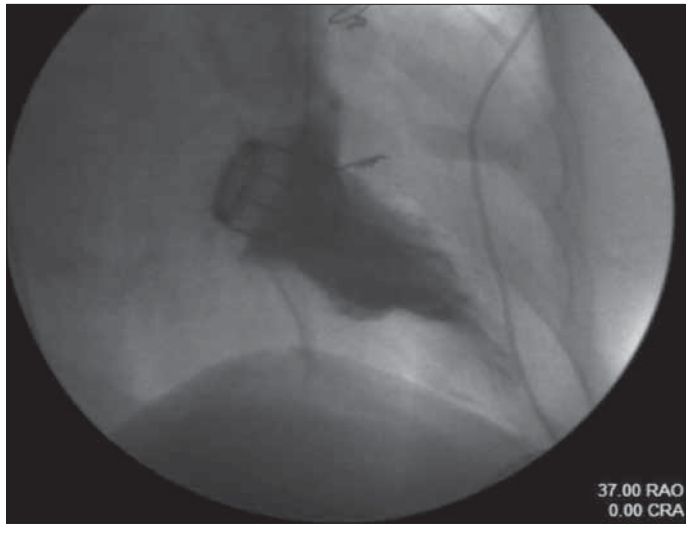

Figure 2. Left ventriculographic view of a Starr-Edwards cage-balled prosthetic mitral valve