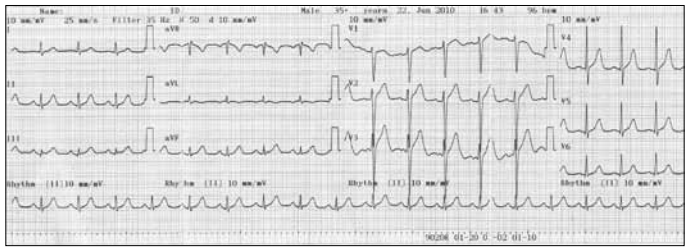

Figure 3. Electrocardiogram after propafenone infusion revealing normal sinus rhythm

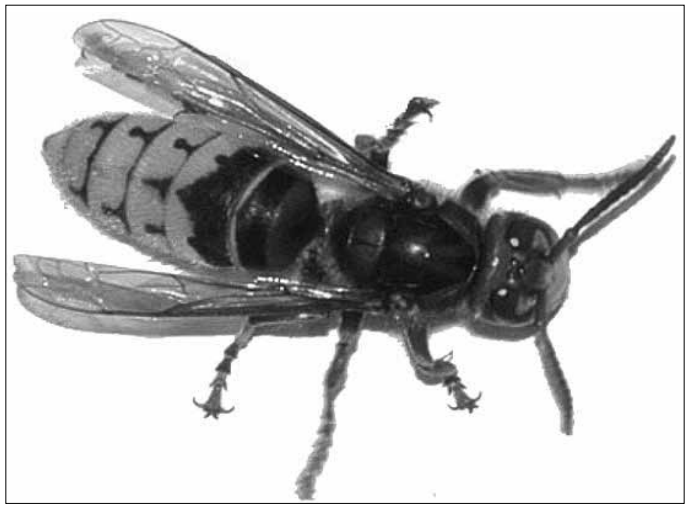

Figure 4. A similar insect was a European hornet (*Vespa Crabro Linnaeus*)

Conclusion

Hornet stings have been associated with a wide variety of local and systemic reactions including atrial fibrillation episodes. Clinical condition is usually self-limiting; electrical cardioversion and/or propafenone are successful therapeutic options.

Video 1. A rare cause of atrial fibrillation: a European hornet sting

References

1. Antonicelli L, Bilo MB, Napoli G, Farabollini B, Bonifazi F. European hornet (Vespa crabro) sting: a new risk factor for life-threatening reaction in hymenoptera allergic patients? Eur Ann Allergy Clin Immunol 2003; 35: 199-203.
2. Fisher BA, Antonios TF. Atrial flutter following a wasp sting. J Postgrad Med 2003; 49: 254-5.
3. Ferrari S, Pietroiusti A, Galanti A, Compagnucci M, Fontana L. Paroxysmal atrial fibrillation after insect sting. J Allergy Clin Immunol 1996; 98: 759-61. [CrossRef]
4. Law DA, Beto RJ, Dulaney J, Jain AC, Lobban JH, Schmidt SB. Atrial flutter and fibrillation following bee stings. Am J Cardiol 1997; 80: 1255. [CrossRef]
5. Jones E, Joy M. Acute myocardial infarction after a wasp sting. Br Heart J 1988; 59: 506-8. [CrossRef]
6. Wagdi P, Mehan VK, Bürgi H, Salzmann C. Acute myocardial infarction after wasp stings in a patient with normal coronary arteries. Am Heart J 1994; 128: 820-3. [CrossRef]
7. Greif M, Pohl T, Oversohl N, Reithmann C, Steinbeck G, Becker A. Acute stent thrombosis in a sirolimus eluting stent after wasp sting causing acute myocardial infarction: a case report. Cases J 2009; 2: 7800. [CrossRef]
8. Vetter RS, Visscher PK. Bites and stings of medically important venomous arthropods. Int J Dermatol 1998; 37: 481-96. [CrossRef]
9. Tisdale JE, Patel RV, Webb CR, Borzak S, Zarowitz BJ. Proarrhythmic effects of intravenous vasopressors. Ann Pharmacother 1995; 29: 269-81. [CrossRef]

Address for Correspondence/Yazışma Adresi: Dr. Sercan Okutucu
Department of Cardiology, Faculty of Medicine, Hacettepe University, Ankara-*Turkey*
Phone: +90 312 305 17 81 Fax: +90 312 311 40 58 E-mail: sercanokutucu@yahoo.com
Available Online Date/Çevrimiçi Yayın Tarihi: 11.08.2011

doi:10.5152/akd.2011.144

Moxifloxacin-dependent *Torsades de Pointes*

Moksifloksasin'e bağlı Torsades de Pointes

Selma Kenar Tiryakioğlu, Osman Tiryakioğlu[1], Faruk Aktürk, Ertuğrul Mehmetoğlu[2], Ethem Kumbay[2]
Clinic of Cardiology, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul
[1]Clinic of Cardiovascular Surgery, Bursa Yüksek İhtisas Training and Research Hospital, Bursa
[2]Clinic of Cardiology, Bursa Acıbadem Hospital, Bursa-*Turkey*

Introduction

Prolongation of the QT interval is a rarely seen side effect of moxifloxacin, and in severe cases it may trigger fatal arrhythmias such as *Torsades de Pointes* (TdP) (1, 2).

"QT prolongation" is a finding that is detected in the surface electrocardiogram and occurs because of the prolongation of the repolarization phase. It may be congenital or acquired. Majority of acquired "long QT" cases are caused by drugs (1, 3).

Case Report

A 56-year-old female was consulted at the emergency service with the complaints of fatigue, presyncope, and dyspnea. Complete atrioventricular (AV) block rhythm was present on the electrocardiogram (ECG). The ventricular rate was 42 beats/min. The QT interval was measured as 468 msec (Fig. 1). The corrected QT (cQT), calculated using Bazett's formula, was 390 msec. Blood pressure was measured as 120/60 mmHg. On physical examination, no cervical venous distension was present. No breath sounds could be heard in either of the lower pulmonary zones. Rales were present, especially in the right lung. On the examination of the cardiovascular system, a 2/6 systolic murmur was detected in the mitral focus. No significant abnormalities were observed in the abdominal examination. Peripheral pulses were palpable. The echocardiographic examination revealed that the left ventricular functions were within normal limits, a prolapsus of the posterior mitral valve was present, and a mild mitral insufficiency flow extending to the anterior region existed. Other echocardiographic parameters were normal. Bilateral pleural effusion was present on the postero-anterior chest X-ray. The patient was not receiving any regular medication. The clinical laboratory findings at the time of admission were as follows: the serum aspartate-amino transferase level was 29 U/L, the serum alanine transaminase

level was 25 U/L, the serum sodium level was 140 mmol/L, the serum potassium level was 4.21 mmol/L, the serum urea nitrogen level was 27 mg/dl, and the serum creatinine level was 0.71 mg/dL. Thus, renal dysfunction, hepatic dysfunction and hypokalemia were not observed during admission. Other biochemical tests were normal.

Due to the patient's stable hemodynamics and sufficient intrinsic cardiac rate, it was decided that the patient did not need a pacemaker but would be closely followed. The patient was started on moxifloxacin because of fever and the high values of erythrocyte sedimentation rate and C-reactive protein, signs that suggested the pulmonary focus. Corrected QT duration was 390 msec on the ECG that was performed during this period. No episodes of profound bradycardia were recorded. On the 5th day of the moxifloxacin treatment, TdP developed (Fig. 2). Rhythm was maintained by electrical cardioversion. On the acquired ECGs, the QT interval duration was 598 msec, whereas the cQT interval - 498 msec (Fig. 3). The patient's heart rate was 39 beat/min. No electrolyte anomalies were present, and the administration of moxifloxacin was discontinued. In the following days, the progressive decrease in QT interval duration was observed on the consecutive ECGs. A DDDR-type pacemaker was implanted to the patient with complete AV block rhythm.

Discussion

Clinically, fluoroquinolones are fairly important antibiotics. They have generally similar safety profiles as other antibiotics and are tolerated well. However, they may have serious side effects (1, 2). One of the side effects of fluoroquinolones is QT interval prolongation, which may cause TdP (3, 4). Moxifloxacin is a third-generation fluoroquinolone that has a wide spectrum, including gram-negative and gram-positive bacteria, anaerobes, and atypical pneumonia agents. It has been demonstrated that moxifloxacin causes less TdP than other fluoroquinolones.

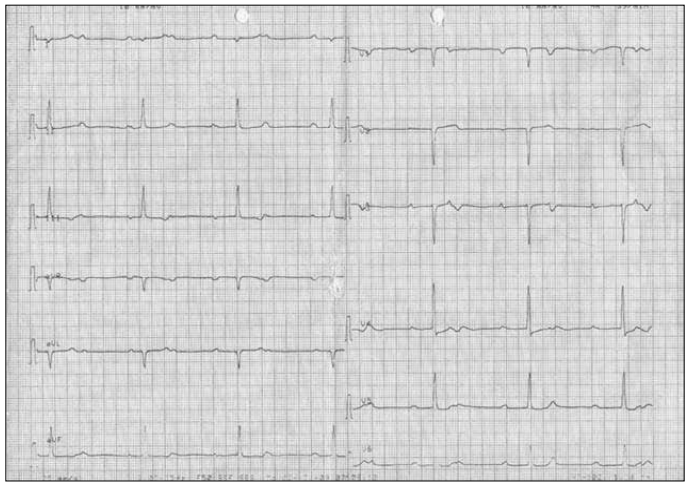

Figure 1. The ECG of the patient on admission. Complete AV block with a rate of 39/min, QT interval duration of 488 msec and corrected QT interval duration of 390 msec are seen (the paper speed was 25 mm/s)
ECG - electrocardiogram

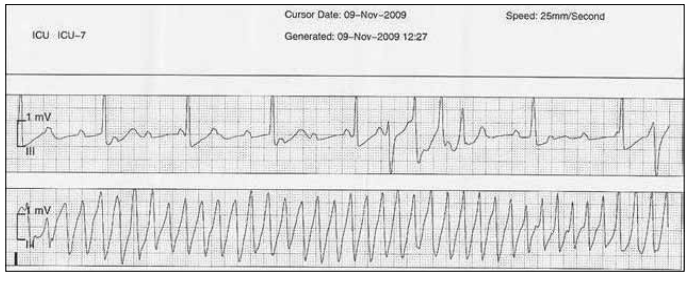

Figure 2. Telemetry recording showing *Torsades de Pointes* (the paper speed was 25 mm/s)

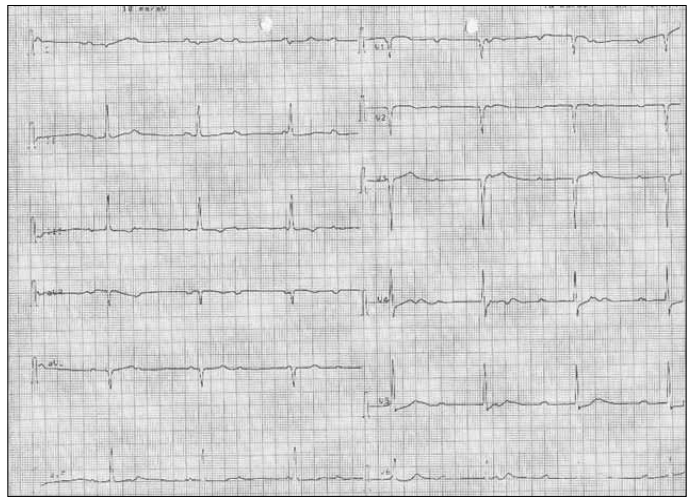

Figure 3. The ECG of the patient after the electrical cardioversion (5th day of moxifloxacin administration). The QT interval duration is 598 msec, whereas the cQT interval duration - 498 msec (the paper speed was 25 mm/s)
ECG - electrocardiogram

Moxifloxacin, like other medications that give rise to long QT syndrome, causes QT interval prolongation as a result of the blockage of the rapid component of delayed rectifier potassium channels (IKr) (5, 6). IKr inhibition delays repolarization by blocking the potassium in myocytes. The blockage of these channels and QT prolongation are dose-dependent. QT interval prolongation is more commonly seen in females, people with organic cardiac disease at an advanced age, people with bradycardia, people with long QT syndrome histories, patients with renal and hepatic dysfunction and people with electrolyte abnormalities (6, 7).

In low cardiac rates, a lesser amount of potassium is released outside the cell due to reduced cardiac repolarization and extracellular potassium concentration. IKr inhibition is inversely correlated with extracellular potassium level. The decrease in potassium concentration increases the IKr blockage level. Thus, the usage of moxifloxacin provides a basis for the development of TdP, especially in patients with bradycardia (7, 8). In the current case, accompanying risk factors (e.g., female gender and advanced age) were also present, which increased the likelihood of developing TdP. However, the QT interval duration observed on the arrival of the patient was within the normal limits. On the 5th day of the moxifloxacin treatment, TdP developed with accompanying serious QT prolongation. TdP might develop in long-term bradycardias due to electrical remodeling. However, we did not consider the bradycardia as long-lasted because the former ECGs of the patient showed sinus rhythm and with normal heart rate. In the consecutive ECGs that were performed, we detected an apparent QT prolongation on the 5th day of moxifloxacin usage. Therefore, we can conclude that moxifloxacin, together with other promoting causes, prolonged QT and resulted in TdP.

Conclusion

If the use of fluoroquinolone group antibiotics is required, patients with multiple risk factors (e.g., advanced age, female sex, severe bradycardia, renal insufficiency) should be followed-up especially carefully

in order to minimize the possibility of QT prolongation. Due to the severe side effects and clinical results of fluoroquinolones, alternative medications should be used if possible.

References

1. Altın T, Özcan O, Turhan S, Ongun Özdemir A, Akyürek O, Karaoğuz O, Güldal M. Torsades de pointes associated with moxifloxacin: a rare but potentially fatal adverse event. Can J Cardiol 2007; 23: 907-8.
2. Bertino J Jr, Fish D. The safety profile of the fluoroquinolones. Clin Ther 2000; 22: 798-817.
3. Iannini PB. Quinolone-induced QT interval prolongation: a not-so-unexpected class effect. J Antimicrob Chemother 2001; 47: 893-4.
4. Ball P, Stahlmann R, Kubin R, Choudhri S, Owens R. Safety profile of oral and intravenous moxifloxacin: cumulative data from clinical trials and post-marketing studies. Clin Ther 2004; 26: 940-50.
5. Owens RC Jr. Risk assessment for antimicrobial agent-induced QTc interval prolongation and torsades de pointes. Pharmacotherapy 2001; 21: 301-19.
6. Koide T, Shiba M, Tanaka K, Muramatsu M, Ishida S, Kondo Y, et al. Severe QT interval prolongation associated with moxifloxacin: a case report. Cases J 2008; 1: 409.
7. Viskin S, Justo D, Halkin A, Zeltser D. Long QT syndrome caused by noncardiac drugs. Prog Cardiovasc Dis 2003; 45: 415-27.
8. Yang T, Roden DM. Extracellular potassium modulation of drug block of IKr. Implications for torsades de pointes and reverse use-dependence. Circulation 1996; 93: 407-11.

Address for Correspondence/Yazışma Adresi: Dr. Osman Tiryakioğlu
Clinic of Cardiovascular Surgery, Bursa Yüksek İhtisas Training and Research Hospital, Bursa-*Turkey*
Phone: +90 224 360 50 50 Fax: +90 224 360 50 55
E-mail: osmantiryaki@gmail.com
Available Online Date/Çevrimiçi Yayın Tarihi: 11.08.2011

doi:10.5152/akd.2011.145

Tek doz parasetamol kullanımına bağlı inferiyor miyokart enfarktüsü

An inferior myocardial infarction due to single dose paracetamol use

Hasan Korkmaz, Mehtap Gürger*
Elazığ Eğitim ve Araştırma Hastanesi, Kardiyoloji ve *Acil Tıp Anabilim Dalları, Elazığ-*Türkiye*

Giriş

Parasetamol oldukça etkili ve yan etkileri bakımından güvenli görülen analjezik ve antipiretik bir ajandır. Dispeptik şikayetlere daha az neden olduğundan ve diğer ilaçlarla etkileşim açısından risk oluşturmadığından, parasetamol günlük pratikte çok fazla reçete edilmekte, hastalar tarafından da tercih edilmektedir. Literatürde yüksek miktarda alınan ilaç dozlarının böbrek ve karaciğer toksisitesine neden olduğuna dair olgular bildirilmesine rağmen, tek doz parasetamolün neden olduğu yan etkileri bildiren olguların sayısı oldukça sınırlıdır. Dahası bu yan etkileri kardiyovasküler sistem bozukluklarıyla ilişkilendiren çalışmalar yok denecek kadar az sayıdadır. Sunacağımız bu olguda altı ay arayla iki kez, tek doz parasetamol aldıktan 15 dakika sonra gelişen miyokart enfarktüsü (MI) irdelenecektir.

Olgu Sunumu

Kırk yaşında erkek hasta acil polikliniğimize, yirmi dakika önce başlayan ve sol kola yayılan, şiddetli ve sıkıştırıcı tarzdaki göğüs ağrısı şikayeti ile başvurdu. Özgeçmişinde, hipertansiyonu ve diyabet tanısı bulunmayan hastanın anamnezinde, asetil salisilik aside karşı alerjisi olduğu ve günde 20 adet sigara içtiği, soy geçmişinde ise önemli bir hastalık öyküsünün olmadığı belirlendi. Acil biyokimyasında: Glikoz: 130 mg/dl, laktat dehidrogenaz: 216 U/L, aspartat aminotransferaz: 43 U/L, alanin aminotransferaz: 39 U/L, kreatin kinaz: 122 U/L, kreatin kinaz -MB: 22 U/L, amilaz: 279 U/L, üre: 28 mg/dl, kreatinin: 0.8 mg/dl, Na: 140 mmol/L, K+: 3.5 meq/L, Ca++: 9.5 mg/dl, total bilirübin: 0.51 mg/dL, lökosit: 8390 mikroL (2 saat sonra bakılan değer 15710), hematokrit: 42.8%, hemoglobin: 12.8 gr/dL, trombosit: 175000 mikroL, sedimentasyon hızı: 2 mm/h idi. Hastanın tipik anjinal ağrısının olması üzerine elektrokardiyografi kaydı alındı. Elektrokardiyografisinde (EKG) saptanan DII-DIII ve aVF derivasyonlarında 3 mm'lik ST elevasyonu ile DI ve aVL derivasyonlarında 2 mm'lik ST depresyonu bulguları, inferiyor MI ile uyumluydu (Şekil 1). Kan basıncı 140/90 mmHg ve nabız 50 atım/dk olan hasta koroner yoğun bakıma yatırıldı. Nefes darlığı olmayan hastanın, yoğun anksiyetesi ve ölüm korkusu mevcuttu. Muayenede döküntüsünün bulunmadığı, cildinin oldukça soluk ve terli olduğu tespit edildi. Yakınlarından alınan anamnezde hastanın, ağrıdan 15 dk önce baş ağrısı nedeni ile 500 mg parasetamol tablet içtiği, altı ay öncede 500 mg parasetamol tablet içtikten sonra bu şekilde göğüs ağrısının başladığı ve beş gün kadar yoğun bakımda takip edildiği öğrenildi. Hasta yakınları o dönemde verilen tedaviyi hatırlamadıklarını ancak hastaya koroner anjiyografi (KAG) yapıldığını ve sonucun normal çıktığını ifade etti. Altı ay öncesi kayıtlara ulaşıldığında kliniğin benzer olduğu, ağrıdan kısa bir süre sonra vücutta kaşıntı, ciltte hiperemi, dudaklarda ödem ve nefes darlığı oluştuğu belirlendi. Bu semptomlarla acil polikliniğine başvuran hastanın o dönemde çekilen EKG'si inferiyor MI ile uyumlu bulunmuştu (Şekil 2). O dönemde hastada alerjik reaksiyon geliştiği düşünüldüğünden, 80 mg Prednol (IV) ve 45.5 mg Feniramin (IV) yapılmış, kısa bir süre sonra da ağrıları geçmiş ve ST rezolüsyonu sağlanmıştı. Troponin takiplerinde 12. saatte sınırda pozitifleşme olmuştu. Vital bulguları stabil hale gelen hastaya kontrast alerjisi yönünden test yapıldıktan sonra KAG uygulanmış, sonuç normal koroner anatomi olarak değerlendirilmişti.

Hasta koroner yoğun bakıma alındı. Bu esnada ciltte hiperemi ve dudaklarda şişme başladı. Bu durumunda alerjik olabileceği ihtimalini düşündüğümüzden trombolitik yerine, 80 mg prednizolon (IV) ve 45.5 mg Feniramin (IV) tedavisi uyguladık. Bu tedaviden beş dakika sonra göğüs ağrısı geçen hastanın, kontrol EKG'sinde ST elevasyonunun düzeldiği ve Q dalgasının oluşmadığı belirlendi (Şekil 3). Takiplerinde stabil hale gelen hastanın, üçüncü ve altıncı saatte troponini negatif, 12. saatte istenen troponin sınırda pozitif geldi. Kullanılan ilacın son kullanma tarihi kontrol edilip sorun olmadığı belirlendi. Hasta stabilleştikten sonra yapılan ekokardiyografisinde ejeksiyon fraksiyonu %60, duvar kalınlıkları, kalp boşluk çapları ve duvar kontraksiyonları normal olarak değerlendirildi. Yapılan efor testi normal olarak değerlendirildi. Hastanemizde immünoglobulin E ve triptaz düzeyleri çalışılamadığından ve alerji testleri yapılamadığından hastanın uygun bir merkeze başvurması önerilerek taburcu edildi.