

ne alındığında, hastaların inflamasyon parametrelerinin preoperatif ya da postoperatif dönemde özellikle değerlendirilmediği de gözlenmektedir (4, 5). Bu çalışmadaki orijinal amacımız; aynı türde cerrahiye maruz kalan hastalarda çeşitli ilaç gruplarının etkinliğini aynı çalışmada araştırılması idi. Yazarların mektubunda belirttiği gibi, AF'u oluşturan tek bir parametreden yola çıkılmamıştır. Şartları genel olarak eşitlenen hasta gruplarında ilaç etkinliği değerlendirilmiş ve bu eşitliği bozacak hastalar dışlanmıştır.

Aynı zamanda bu çalışmada amaç gen polimorfizmi olan hastalarda ilaç etkinliklerinin farklı olup olmayacağını araştırmak da değildir. Gerçekten çok ilginç olan bu konuda çok sayıda çalışma yapılabileceği görüşündeyiz.

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Kaynaklar

1. Men EE, Yıldırım Ö, Tuğcu A, Aytekin V, Aytekin S. Açık kalp cerrahisi sonrasında gelişen atriyal fibrilasyonu önlemek için kullanılan ilaçların etkinlik yönünden karşılaştırılması. Anadolu Kardiyol Derg 2008; 8: 206-12.
2. Isaac TT, Dokainish H, Lakkis NM. Role of inflammation in initiation and perpetuation of atrial fibrillation. J Am Coll Cardiol 2007; 50: 2021-8.
3. Omen Sr, Odell SA, Stanton MS. Atrial arrhythmias after cardiothoracic surgery. N Engl J Med 1997; 337: 1785-90.
4. Tisdale JE, Pahdi ID, Goldberg AD, Silverman NA, Webb CR, Higgins RS, et al. A randomized, double-blind comparison of intravenous diltiazem and digoxin for atrial fibrillation after coronary artery bypass. Am Heart J 1998; 135: 739-47.
5. Paul DL, Tidwell SL, Guyton SW, Harvey E, Woolf RA, Holmes JR, et al. Beta blockade to prevent atrial dysrhythmias following coronary bypass surgery. Am J Surg 1997; 173: 419-21.

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Assessment of aortic stiffness and ventricular functions in familial Mediterranean fever

Ailevi Akdeniz ateşinde aortik sertleşme parametrelerinin ve ventrikül fonksiyonlarının değerlendirilmesi

Dear Editor,

Familial Mediterranean fever (FMF) is an autosomal recessive disorder virtually restricted to certain ethnic groups originating from the Middle East: Sephardic Jews, Armenians, Arabs, Druze and Turks (1). It is characterized by recurrent episodes of serosal inflammation, chest pain, and arthritis usually accompanied by fever (1). The main complication of untreated patients is the development of amyloidosis (1). In most FMF patients, colchicine treatment prevents febrile attacks and development of amyloidosis. During the febrile attacks, an acute phase response develops, manifested by a marked increase in erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), serum amyloid A, fibrinogen and leucocytes (1). Histopathologic examination of FMF involves inflammation with infiltration by neutrophils (1). Systemic inflammation is an important factor in the initiation or the progression of atherosclerosis. Damage to the arterial wall due to inflammation and atherosclerosis causes decreased arterial distensibility, compliance and elasticity (2-4). Non-invasive ultrasound

techniques are used to evaluate vascular system and cardiovascular condition (3, 4). One such technique, Doppler pulse wave velocity (PWV), which is defined as arterial pulse's velocity of moving along vessel wall, as an indicator of arterial elasticity (2-4). Pulse wave velocity is inversely correlated with arterial distensibility and relative arterial compliance. Inflammation may play a role in the process of arterial stiffening (3, 4).

We read with interest the article "Assessment of aortic stiffness and ventricular functions in familial Mediterranean fever" by Sari et al. (5) which compared the aortic stiffness and ventricular functions in patients with FMF and control group. The authors have reported the aortic wall properties were similar between two groups, however, we have recently showed that the carotid-femoral PWV was slightly higher in colchicine-treated FMF patients than in control subjects ($p=0.05$) (4). We also found significant correlation between PWV and age ($p<0.001$, $r=0.67$), body mass index ($p<0.001$, $r=0.52$) and leucocytes ($p<0.001$, $r=0.66$) in all groups and in patients with FMF group ($p<0.001$, $r=0.73$; $p=0.01$, $r=0.52$; $p<0.001$, $r=0.69$, respectively) (4). The inflammatory process of FMF may act to impair endothelial function, arterial compliance and arterial elasticity and as a contributing factor in the initiation or the progression of atherosclerosis. In the light of these findings, we think that Sari et al. should detail why the aortic elastic properties and pericardium showed no significant difference between patients with FMF group and healthy controls groups.

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References

1. Sohar E, Gafni J, Pras M, Heller H. Familial Mediterranean fever. A survey of 470 cases and review of the literature. Am J Med 1967; 43: 227-53.
2. Yıldız M, Sahin B, Sahin A. Acute effects of oral melatonin administration on arterial distensibility, as determined by carotid-femoral pulse wave velocity, in healthy young men. Exp Clin Cardiol 2006; 11: 311-3.
3. Yıldız M, Soy M, Kürüm T, Yıldız BS. Arterial distensibility in Wegener's granulomatosis: a carotid-femoral pulse wave velocity study. Anadolu Kardiyol Derg 2007; 7: 281-5.
4. Yıldız M, Masatlıoğlu S, Seymen P, Aytac E, Sahin B, Seymen HO. The carotid-femoral (aortic) pulse wave velocity as a marker of arterial stiffness in familial Mediterranean fever. Can J Cardiol 2006; 22: 1127-31.
5. Sari I, Arican O, Can G, Akdeniz B, Akar S, Birlik M, et al. Assessment of aortic stiffness and ventricular functions in familial Mediterranean fever. Anadolu Kardiyol Derg 2008; 8: 271-8.

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Author's Reply

Dear Sir,

We thank authors for their interest and valuable comments on our recent publication (1).

Familial Mediterranean fever (FMF) is an auto-inflammatory rheumatic disease characterized by periodic attacks of fever and serositis. During the attack free periods, subclinical inflammation continues (2). In recent years markers of increased early atherosclerosis have been reported in various inflammatory rheumatic diseases including FMF (1). On the other hand, increase in aortic stiffness is a manifestation of vascular damage and predictor of cardiovascular mortality. Thus, measurement of arterial stiffness became an important part of risk assessment and monitoring the efficacy of therapy in patients with conditions such as isolated systolic

hypertension (3). At present, there are several methods available that can be used to analyze arterial elasticity. Although invasive methods remain gold standard, noninvasive techniques are widely used in clinical settings as these methods give us safe and accurate means of detecting of arterial elasticity. Among them, pulse pressure, pulse wave velocity, ultrasound derived indices, waveform analysis and magnetic resonance imaging derived indices are the most commonly used and popular methods (4, 5).

The study by Yıldız et al. used carotid and femoral Doppler pulse wave velocity (PWV) and in the present study, we estimated aortic distensibility from echocardiographic measurements of aortic diameter at systole and diastole, and aortic pressure was assessed by brachial cuff blood pressure taken at the time when echocardiographic measurements were made. This method enables us to estimate the elastic properties of the ascending aorta from its direct measurements. Although carotid and femoral PWV requires little technical expertise and used widely, ultrasound derived methods are also reliable and used in clinical settings extensively (6, 7).

The former study by Yıldız et al included 23 FMF patients and controls and according to their results, although missed significance, PWV was slightly higher in FMF group (8). In contrast, our results were not different between patients and controls. Although both groups had similar age ratios and body composition parameters (Table 1), mainly two important factors might be responsible from this situation: 1- methodological differences may be accounted from the condition, and 2- as figured out from the high mean C- reactive protein values in the group of patients studied by Yıldız et al., higher inflammatory burden might affect the results.

In conclusion, further studies comprising new promising techniques such as MRI and studies including active and inactive FMF patients are needed to determine whether aortic stiffness in FMF is increased or not.

Table 1. Some demographical and laboratory findings of studies conducted by Sari et al (1) and Yıldız et al (8)

	Study by San et al.		Study by Yıldız et al.	
	FMF	Controls	FMF	Controls
Number of subjects	44	27	23	23
Sex, M/F	21/23	12/15	6/17	6/17
Age, years	32.6±9.2	30.9±4.7	29.4±8.7	29.2±9
BMI, kg/m ²	24.7±4.1	24.5±3.8	23.29±3.53	23.47±4.1
WHR	0.84±0.08	0.82±0.09	0.82	0.80
Mean blood Pressure, mm/Hg	88.7±8.9	90.8±6.8	77.75±9.26	81.87±7.98
Fasting glucose, mg/dL	85.4±6.1	83.3±7.4	-	-
Total cholesterol, mg/dL	162±31.8	170±30.2	166.86±36.64	163±27.38
LDL cholesterol, mg/dL	92±29.6	95±25.4	103.73±26.94	90.85±26.02
HDL cholesterol, mg/dL	50.3±10.8	56.4±14.5	-	-
Triglyceride, mg/dL	108±43.3	90±35.8	99.3±39.19	100±28.17
ESR, mm/h	17.7±17.9	8.9±5.3	16.65±11.97	10.00±1.63
CRP, mg/dL	0.67±1.23	0.17±0.21	1.35±2.26	0.27±0.11

BMI - body mass index, CRP - C-reactive protein, ESR - erythrocyte sedimentation rate, F- female, HDL - high density lipoprotein, LDL - low density lipoprotein, WHR - waist-hip ratio, M- male

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References

1. Sari I, Arican O, Can G, Akdeniz B, Akar S, Birlik M, et al. Assessment of aortic stiffness and ventricular functions in familial Mediterranean fever. *Anadolu Kardiyol Derg* 2008; 8: 271-8.
2. Onen F. Familial Mediterranean fever. *Rheumatol Int* 2006; 26: 489-96.3. Boutouyrie P, Laurent S, Briet M. Importance of arterial stiffness as cardiovascular risk factor for future development of new type of drugs. *Fundam Clin Pharmacol* 2008; 22: 241-6.
4. Mackenzie IS, Wilkinson IB, Cockcroft JR. Assessment of arterial stiffness in clinical practice. *QJM* 2002; 95: 67-74.
5. Boutouyrie P. New techniques for assessing arterial stiffness. *Diabetes Metab.* 2008;34 Suppl 1: S21-6.
6. Marcus RH, Korcarz C, McCray G, Neumann A, Murphy M, Borow K, et al. Noninvasive method for determination of arterial compliance using Doppler echocardiography and subclavian pulse tracings. Validation and clinical application of a physiological model of the circulation. *Circulation* 1994; 89: 2688-99.
7. Stefanadis C, Stratos C, Boudoulas H, Kourouklis C, Toutouzas P. Distensibility of the ascending aorta: comparison of invasive and non-invasive techniques in healthy men and in men with coronary artery disease. *Eur Heart J* 1990; 11: 990-6.
8. Yıldız M, Masatlioglu S, Seymen P, Aytac E, Sahin B, Seymen HO. The carotid-femoral (aortic) pulse wave velocity as a marker of arterial stiffness in familial Mediterranean fever. *Can J Cardiol* 2006; 22: 1127-31.

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Kemik iliği transplantasyonu sırasında kullanılan yüksek doz siklofosfamide bağlı inferiyor miyokard infarktüsünü taklit eden vazospastik angina olgusu

Vasospastic angina mimicking inferior myocardial infarction due to high dose cyclophosphamide for bone marrow transplantation conditioning

Kanser tedavisinde kullanılan antrasiklinler, paklitaksel, trastuzumab, siklofosfamid ve 5-fluorouracil kardiyak açıdan toksik kemoterapötik ajanlardır (1). Alkilye bir ajan olan siklofosfamid sıklıkla kemik iliği nakli sırasında yüksek dozlarda kullanıldığında akut miyoperikardite sebep olabilir (2) ve ortaya çıkan siklofosfamid toksisitesi ölümcül olabilir (3).

Elli altı yaşında bayan hasta, başvurusundan yaklaşık 1.5 ay önce meme kanseri tanısı konulduktan sonra, yapılan tetkiklerinde hemoglobin 7.2 gr/dl, beyazküresi 49680 10³/ul, trombositleri 7600010³/ul saptanması üzerine hematoloji servisine yatırıldı ve akut non-lenfoblastik lösemi-M5 tanısı konuldu. Hasta toplam 135 mg adriablastina ve 28,3 gr sitozin arabinosid tedavisi aldı. Nisan 2007'de allogeneik kök hücre nakli yapıldı. Kemik iliği nakli öncesi yapılan hazırlık tetkiklerinden elektrokardiyogram (EKG) ve ekokardiyografisi (EKO) normal idi. Hazırlama rejimi olarak, total 896 mg busulfan ve 4200 mg siklofosfamid verildi. Siklofosfamid tedavisinden 12 saat sonra çekilen EKG'sinde sinüs ritmi, 105 atım/dk, D2-D3-aVF'de ST elevasyonu, D1-aVL ve V1'den V6'ya kadar ST çökmeleri izlendi. Kardiyak enzimleri normal saptandı. Siklofosfamid tedavisinden 24 saat sonraki EKG'sinde ise ST elevasyon ve çökmelerinin kaybolduğu normal bir EKG izlendi.