

Hipertansiyon ve Hiperlipidemi tedavisine Güncel Yaklaşım

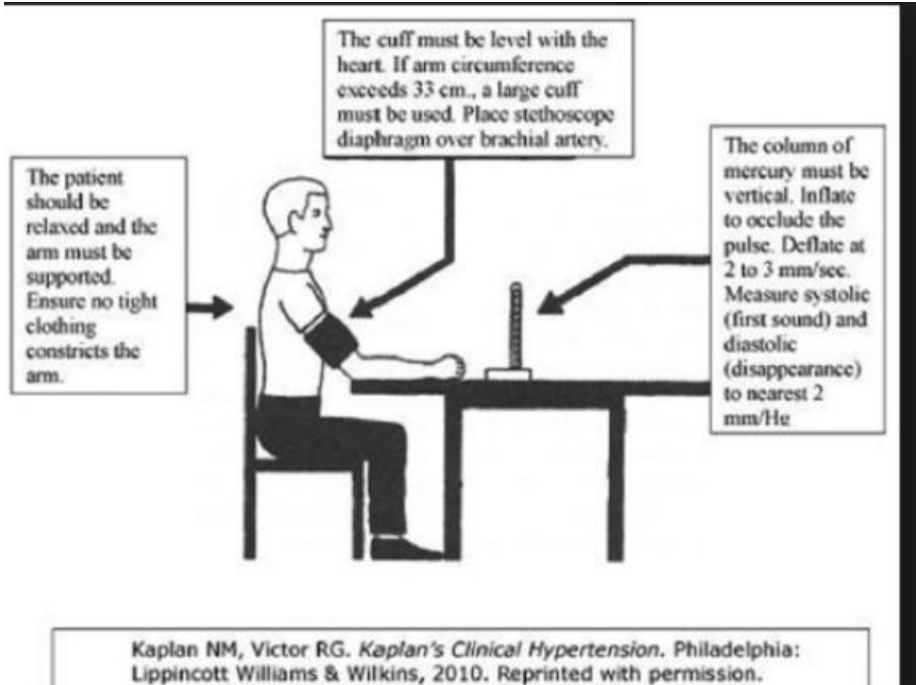
Prof.Dr.Hüseyin Oflaz

Kardiyoloji uzmanı

EGEKON-28.05.2022

Hipertansiyon tanı ve tedavisi (ESC/ESH 2018)

Tansiyonu doğru ölçüyor muyuz?

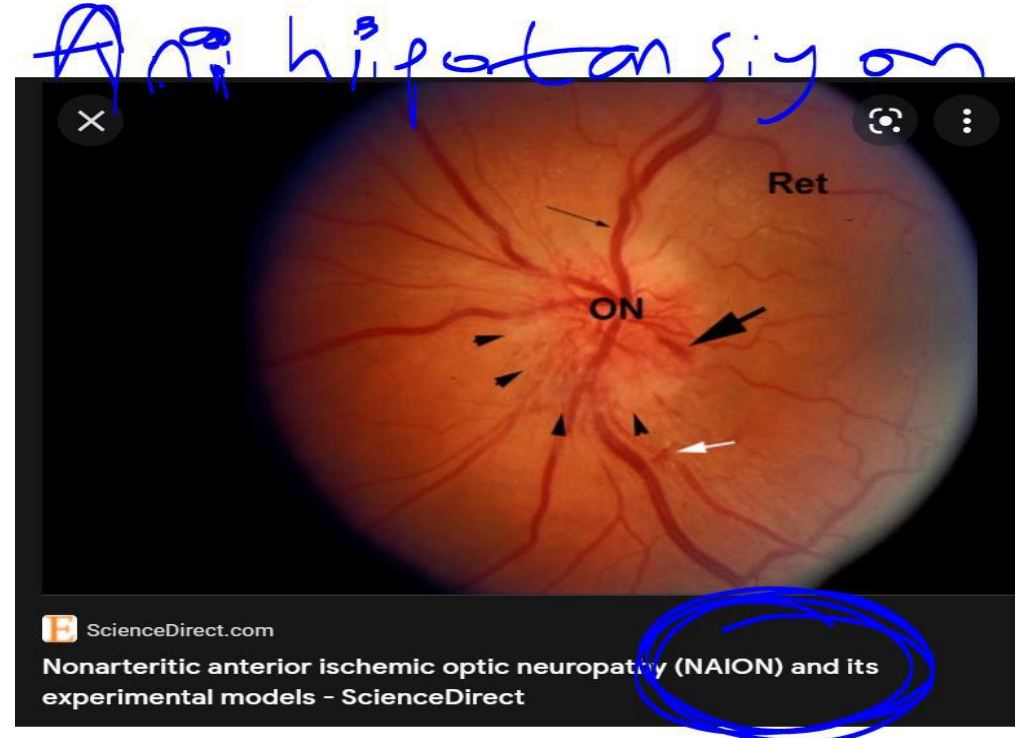


- İngiltere 'de %60 a yakın dr yanlış ölçüyor
- Bizde tansiyon yanlış ölçüm oranı daha az. Çünkü tansiyon ölçülüyor...
- Kılavuzlarda yazan değerler kurallarına göre ölçülmüş olanlara göre ilaç vb..
- Hasta ölçmeden önce sigara, yemek vb 1 saat geçmiş olacak, tuvalet ihtiyacı vb olmayacak, 10-20 dk istirahat sonrası ölçülecek...
- Manşon çevresi, kol rahatlığı, manşonun havasının inme hızı ve kortkoff sesleri...

Doğru tansiyon ölçmek gereksiz ilaç kullanımını önler

Tuvalete sıkışmış teyzede hastanede tansiyon 180/100, evde 120/70 mmHg olabilir

- Doğru tansiyon ölçmenin önemi. Genellikle yanlış yüksek bulunan çok ve gereksiz yere ilaç yazılabiliyor ve düşük tansiyonunda organlara zarar verdiğini bilmeliyiz.
- NAION gözde görme kaybı=
- (iskemik optik nöropati)



Hipotansiyondan da korkalım-DM ve yaşlılarda
otoregülasyon bozulduğundan ani düşme ve yükselme olabilir

- Yanlış ölçüme bağlı yanlış daha yüksek bulunan hastaya verilen gereksiz antihipertansifler hastada ciddi hipotansiyon ve organ hasarı yapabilir
- **Hipertansif acillerde aniden tansiyonu 120-130 mmH getirmek hipoperfüzyona ve ciddi organ iskemisi ve yetersizliklerine yol açabilmekte** (akut böbrek yetersizliği, koroner iskemi, beyinde iskemik inme vb bulgular, gözde NAION(optik iskemik hasar=körlük)

Hipertansiyon tedavisinde başarılı olmak istiyorsanız hastaya hipertansiyonun ne kadar ciddi olduğunu anlatmalıyız

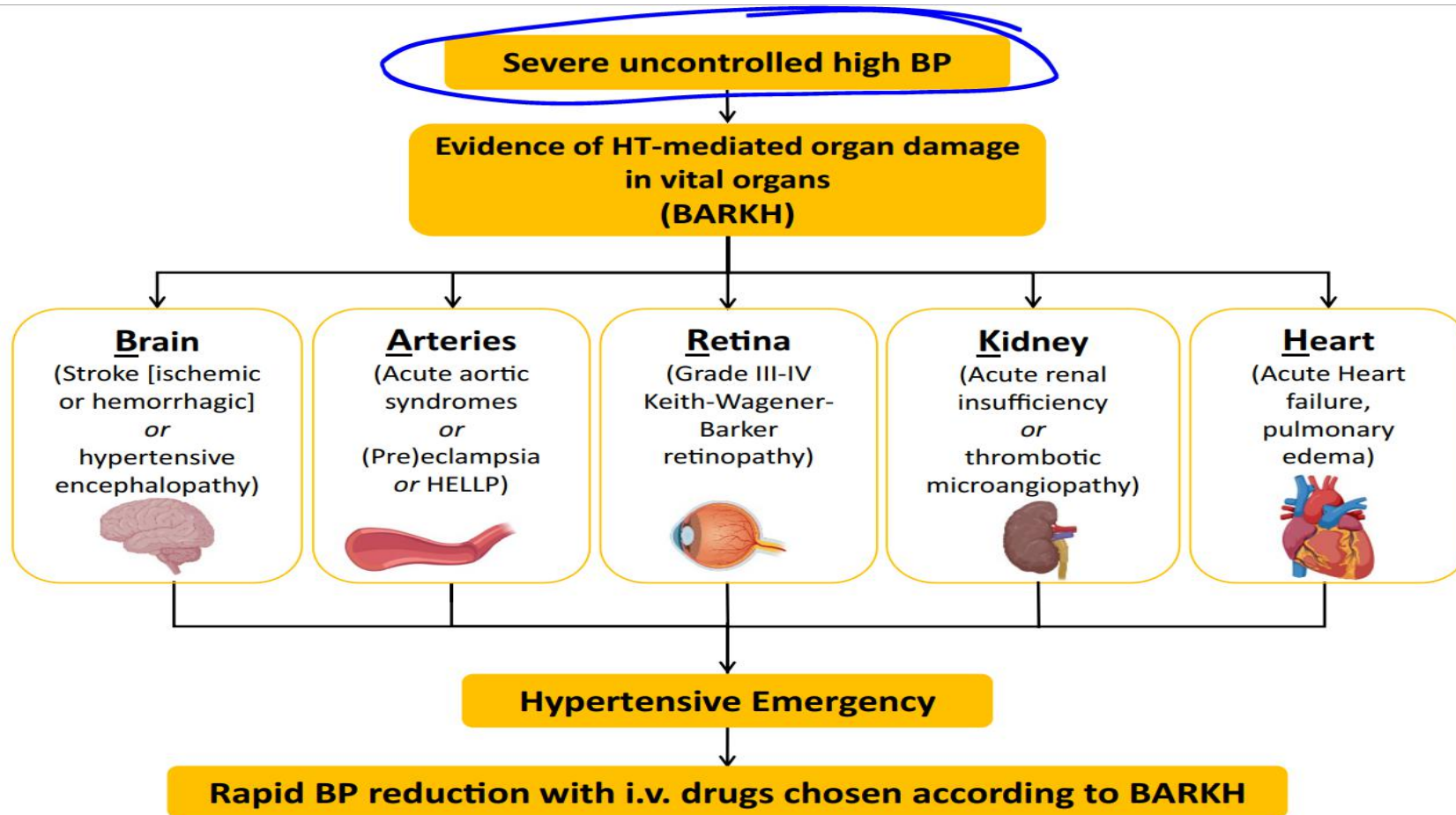


Fig. 1 Brain, arteries, retina, kidney and/or heart (BARKH)-based algorithm. The algorithm allows a quick identification of the hyper-

lack of BARKH involvement rules out the need for i.v. treatment. *BP* blood pressure, *HELLP* haemolysis elevated liver enzymes low plate-

Uyku apneyi soruyor ve ciddiye alıyor muyuz?



- Sekonder hipertansiyon sebepleri arasında obezite ve uyku apne için anemnezde mutlak sorgulamak

Symptoms of Sleep Apnea



- Poor concentration
- Low mood
- Restless sleep
- Heartburn
- Waking up in the morning with a dry mouth or throat
- Waking up with a headache
- Night sweats
- Insomnia
- Waking up dizzy
- Weight gain
- Excessive daytime fatigue
- Lack of energy
- Forgetfulness
- Irritability
- Sexual dysfunction
- Frequent urination at night

Anemnezde nelere dikkat ediyoruz?

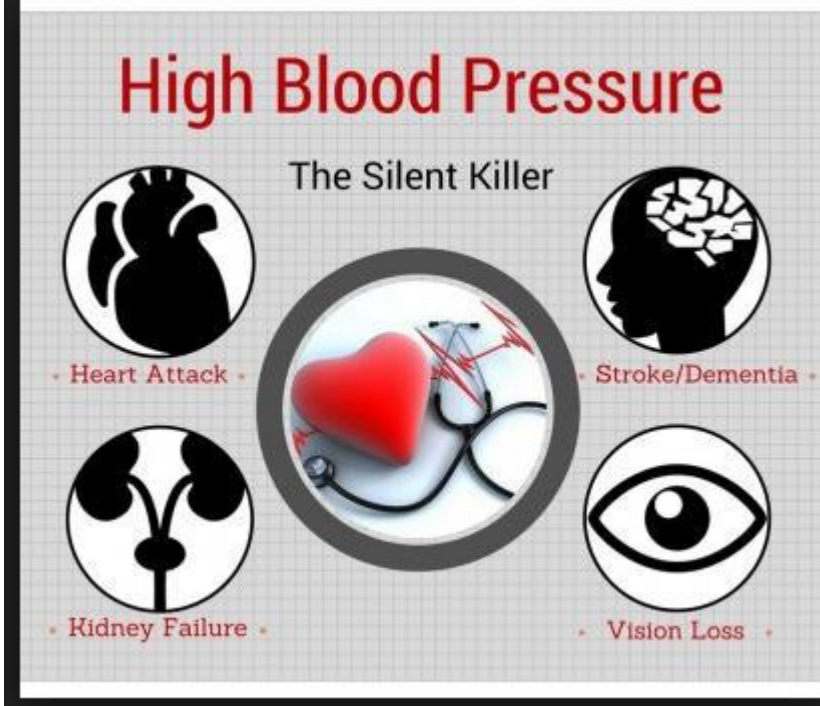
- **Hipertansif hastanın anemnezinde;**
 - Uyku apne,
 - Psödoefedrinli damla ya da tablet, efexor ve cymbalta vb ilaçlar,
 - Gençlerde kokain, enerji içeceği tüketimi vb madde kullanımı, bazı bölgelerde meyan kökü şerbeti vb sorgulanması gerekli

Hipertansiyon= Kanser (HİPERTANSİNOMA)

Hiç abartılı bir benzetme değil

Bir glioblastome multiforme değil ama bir kolon ca kadar tehlikeli olabilir...

Silent killer neden mi?ište size komplikasyonlar



- Hemorajik stroke (SAK ve intraserebral..)
- AF ve bu zeminde iskemik inme
- Myokard infarktüsü
- Ani ölüm
- Diyastolik ky
- Periferik arter hastalığı
- Görme kaybı=Hipertansif retinopati
- Son dönem böbrek yetersizliği
- Demans ve kognitif fonksiyonlarda azalma



Hipertansiyon sınıflaması- bu rakamlar kuralına göre ölçülmüş tansiyon değerleridir

Table 3 Classification of office blood pressure^a and definitions of hypertension grade^b

Category	Systolic (mmHg)		Diastolic (mmHg)
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥180	and/or	≥110
Isolated systolic hypertension ^b	≥140	and	<90

Ofis, ev ve holtere göre hipertansiyon

Table 9 Definitions of hypertension according to office, ambulatory, and home blood pressure levels

Category	SBP (mmHg)		DBP (mmHg)
Office BP ^a	≥140	and/or	≥90
Ambulatory BP			
Daytime (or awake) mean	≥135	and/or	≥85
Night-time (or asleep) mean	≥120	and/or	≥70
24 h mean	≥130	and/or	≥80
Home BP mean	≥135	and/or	≥85

Hipertansiyon tanımları

- **Gerçek patolojik hipertansiyon** (Sustained=Devamlı); hem ofiste (HASTANE yada muayenehane) hemde ev yada holterde tansiyon yüksekliği kriterlerini doldurmuş tansiyon
- **Gerçek normal tansiyon**; Hem ofiste hemde evde hemde holterde tansiyon normal sınırlarda
- **Beyaz gömlek hipertansiyonu**
 - Ofiste tansiyon tanımı alıyor yani 140-90 üstü ama ev veya holterde tansiyon tanımı almıyor...Sonuç; Hipertansiyon yok....
 - Neden hastanede tansiyon yükseliyor dersiniz genelde beyaz gömlekli dr ya da hemşire görmek desek de aslında hastane ortamı vb etkileri var. Şimdi ilaç yazacak mı?, başka hastalık da bulacak mı anksiyetesi?, hastaneden kuyruk ve bürokrasi ve tuvalet vb bulamamak ve uzun beklemek....hepsi tansiyonu yüksek yapar...
- **Maskelenmiş hipertansiyon**
 - Burada ise beyaz gömleğin tam tersi olup ofiste tansiyon normal ama evde veya holterde tansiyon yüksek

Hipertansif hastalarda rutin tetkikler

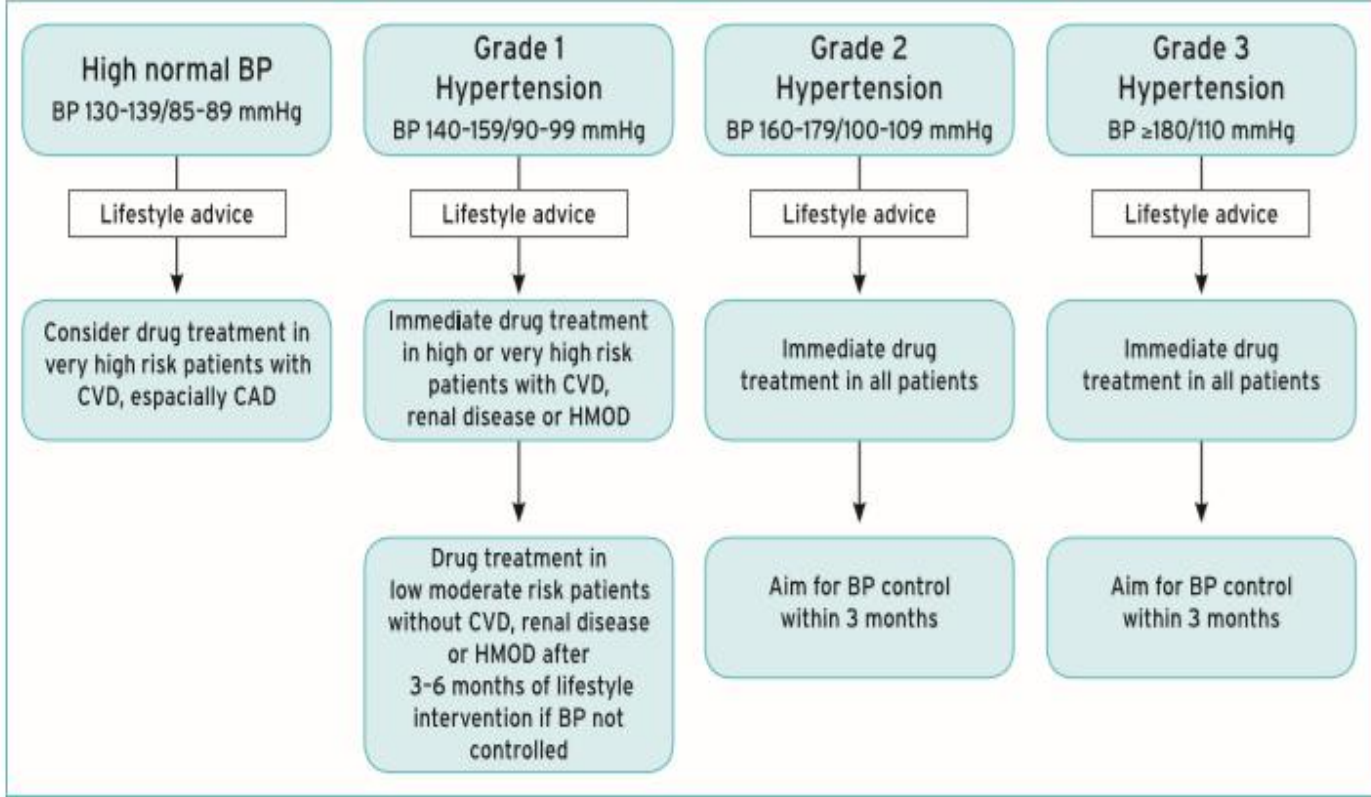
-Hipertansiyon hastasında rutin laboratuvarda d vitaminine duyduğumuz heyecan kadar proteinüri heyecanında duyuyor muyuz?

- Hemogram
- Açlık glikozu ve HbA1c
- Lipidler
- Sodyum ve potasyum
- Ürik asit
- Kreatinin ve GFR
- Karaciğer fonksiyon testleri
- İdrar tetkiki
- İdrarda dipstick ile proteinüri varlığı veya SABAHA SPOT İDRARDA ideali albümin/kreatinin oranı
- Ekg

İdrar tahlilinde proteinüri varsa önemi

- Proteinürisi olanlarda ateroskleroz daha yaygın, ciddi endotel hasarı yapıyor (hasarlı endotelde tromboz ve ateroksleroz sık olur)
- Proteinürisi olan varsa tromboz daha sık (akut Mi, inme veya DVT vb)
- Proteinürinin kendisi nefrotoksik-tubulu-toksik-KBY gidişi hızlandırıyor
- Proteinürisi olan DM hastasında DM sürem 3 yıl diye bilse de bu hasta olası KV hastalık vb risk yönünden **ÇOK YÜKSEK RİSK** grubunda bir DM hastası sayılır
- **En iyi antiproteinürik ilaç ACE-i ve ARB** (KBY ve diyalizlerin önemli bir kısmı hipertansif nefroskleroz ve sizin önceden vereceğiniz ACE-inhib diyalize gidişi önlüyor)

Hipertansiyonda tedavi



***130-139/85-89 (yüksek normal)**

Hedef organ tutulumu (HOT) varsa ACE-i başta olmak üzere ilaç başla, izole HT ise yaşam tarzı değişikliği (YTD) yeter.

Evre -1

HOT yoksa 3-6 YTD, yine düşmezse ilaç ekle. HOT varsa direkt ilaç başla

Evre-2- Direkt ilaç başla

Evre -3- Direkt ilaç başla

Neden ilk tercihimiz diüretikli plus/fort vb yazan antihipertansifler oluyor?

- **Diüretiklerin gereksiz kullanımı** ile artan hiponatremi, hipopotasemi ve ürik asit yüksekliğini ne kadar ciddiye alıyor ve **takip için ilaç sonrası 2 hafta, sonra 3-4 ay da bir kreatinin, sodyum, potasyum, ürik asit bakıyoruz**

Antihipertansif tedavideki başarısızlığı en sık sebebi ilaçların erken kesilmesi (kötü ilaç uyumu)

- Hipertansiyon kanser kadar tehlikeli olabilir, ama ilaç alır, kilo verir ve tuzu azaltırsan korkmanıza gerek yok demeli
- İlaçların kullanımının önemi hastaya vurgulanmalı. Bu ilaçların;
 - Böbreğe ve karaciğer dokunmadığını
 - Ödem ve öksürük yaparsa masum olduğunu, ilaç kesince düzeleceği anlatılmalı
 - İlaçlara 1 gün dahi ara verilince rebound olup hipertansif acil olabileceği (kafa içi kanama, akut akciğer ödemi vb) anlatılmalı

Hipertansiyon tedavisinde başarısızlığın en sık sebebi, ilaç uyunç=uyum bozukluğu

- Hedef hipertansiyon ancak hastaların %30 unda...
- Sebep bu yaşta ilaç kullanılır mı, bağımlılık yapacak, karaciğer ve böbreğimi mahvedecek bunlar..
- Genç hipertansif obezite vb nedeniyle arttı ve ilaç kullanımı gençlerde daha büyük sorun..
- Çok net olarak anlatılırsa tedavide başarı şansınız artar...

Komplike olmamış hipertansiyonda ilk tedavi

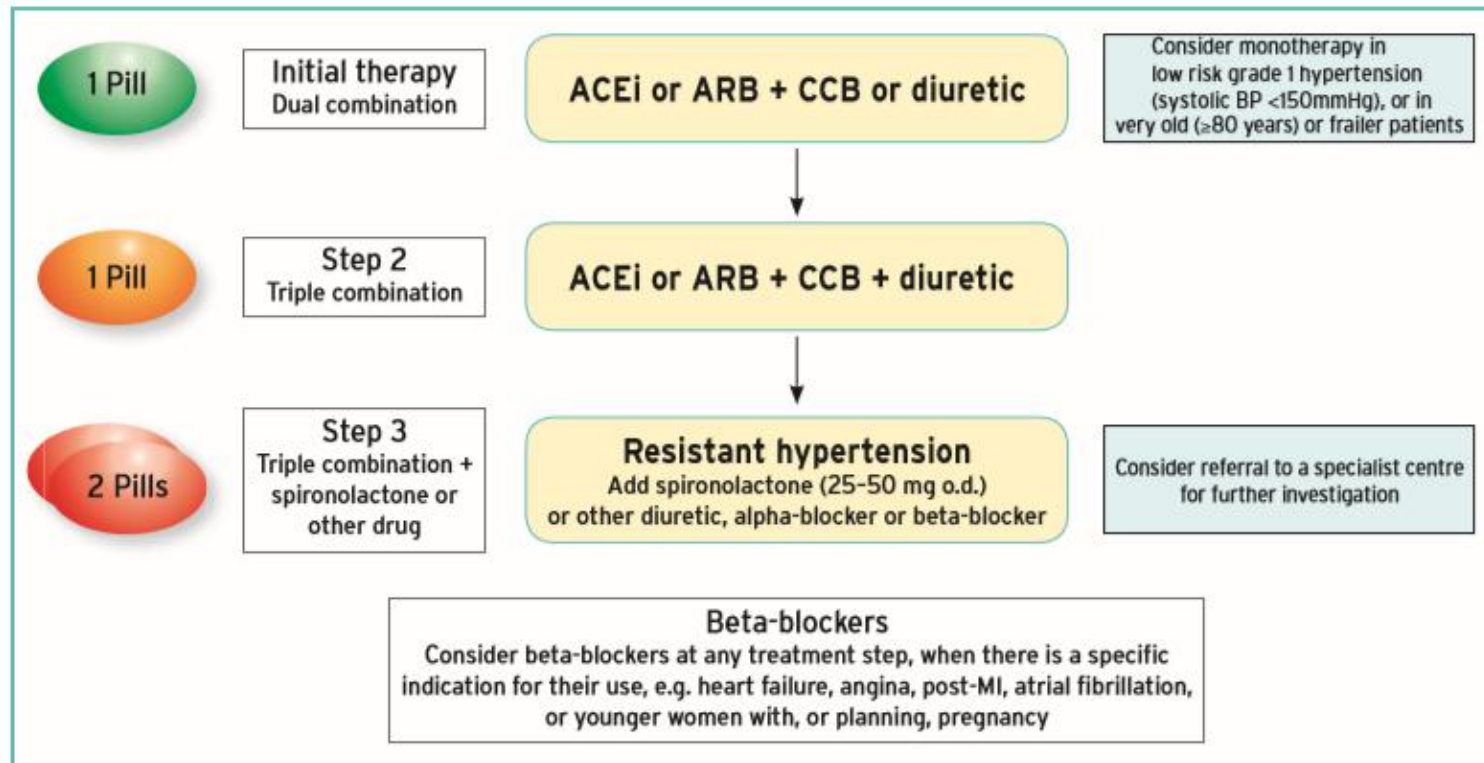


Figure 4 Core drug treatment strategy for uncomplicated hypertension. The core algorithm is also appropriate for most patients with

Koroner arter hastası olanda hipertansiyon tedavisi

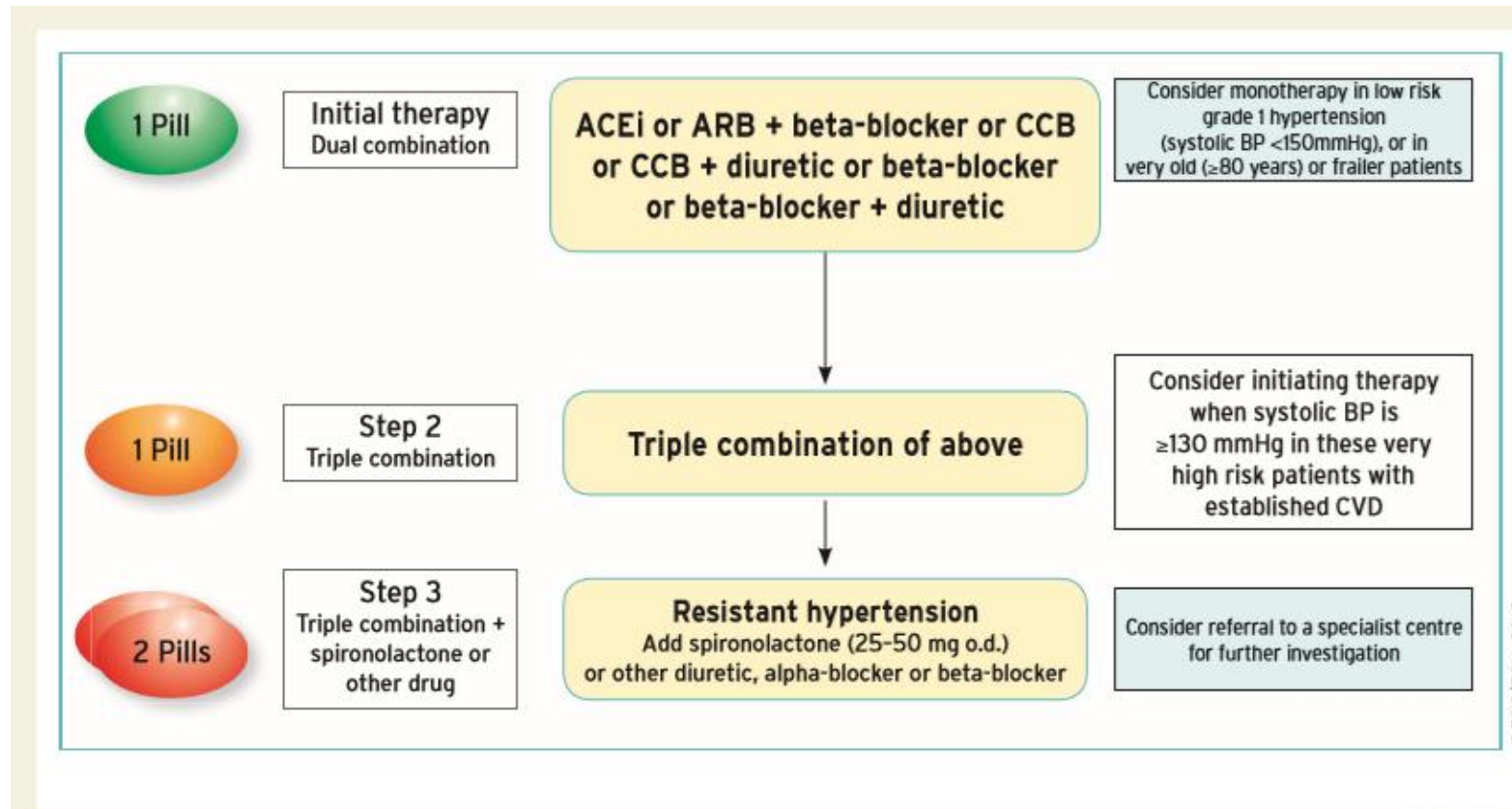
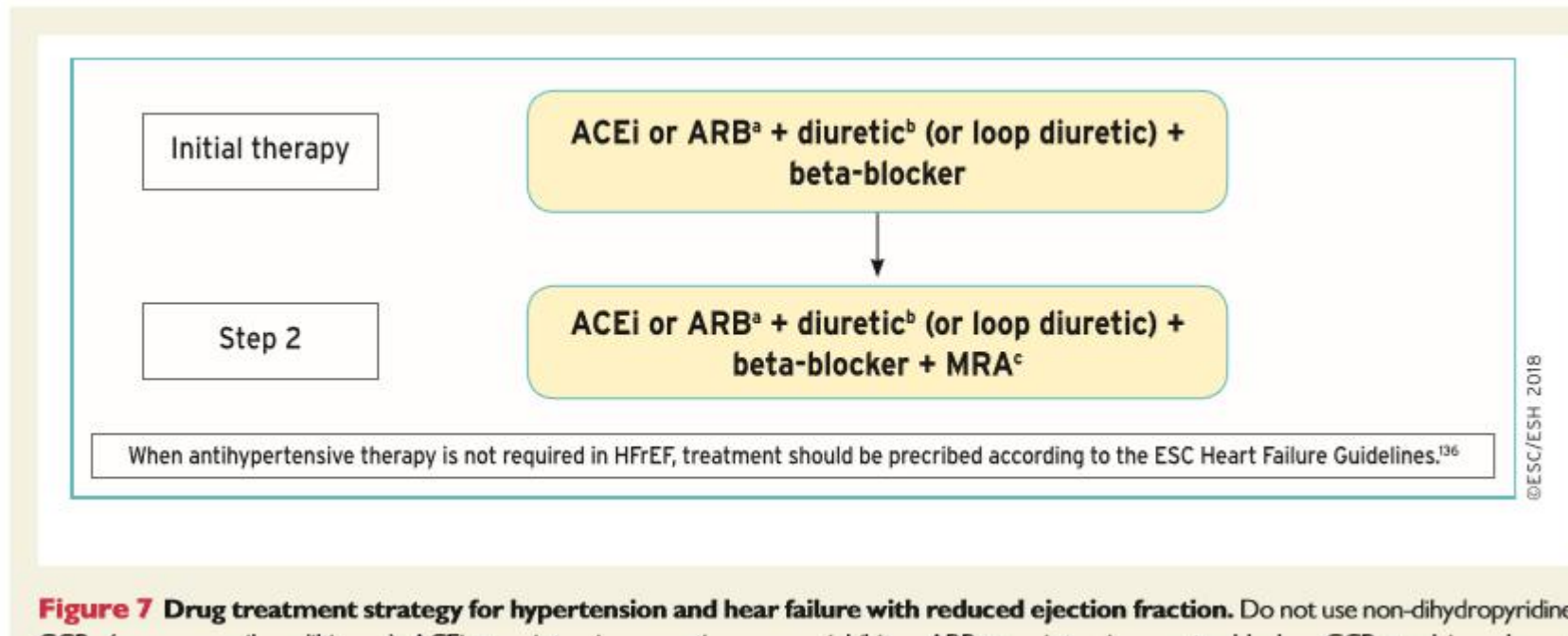
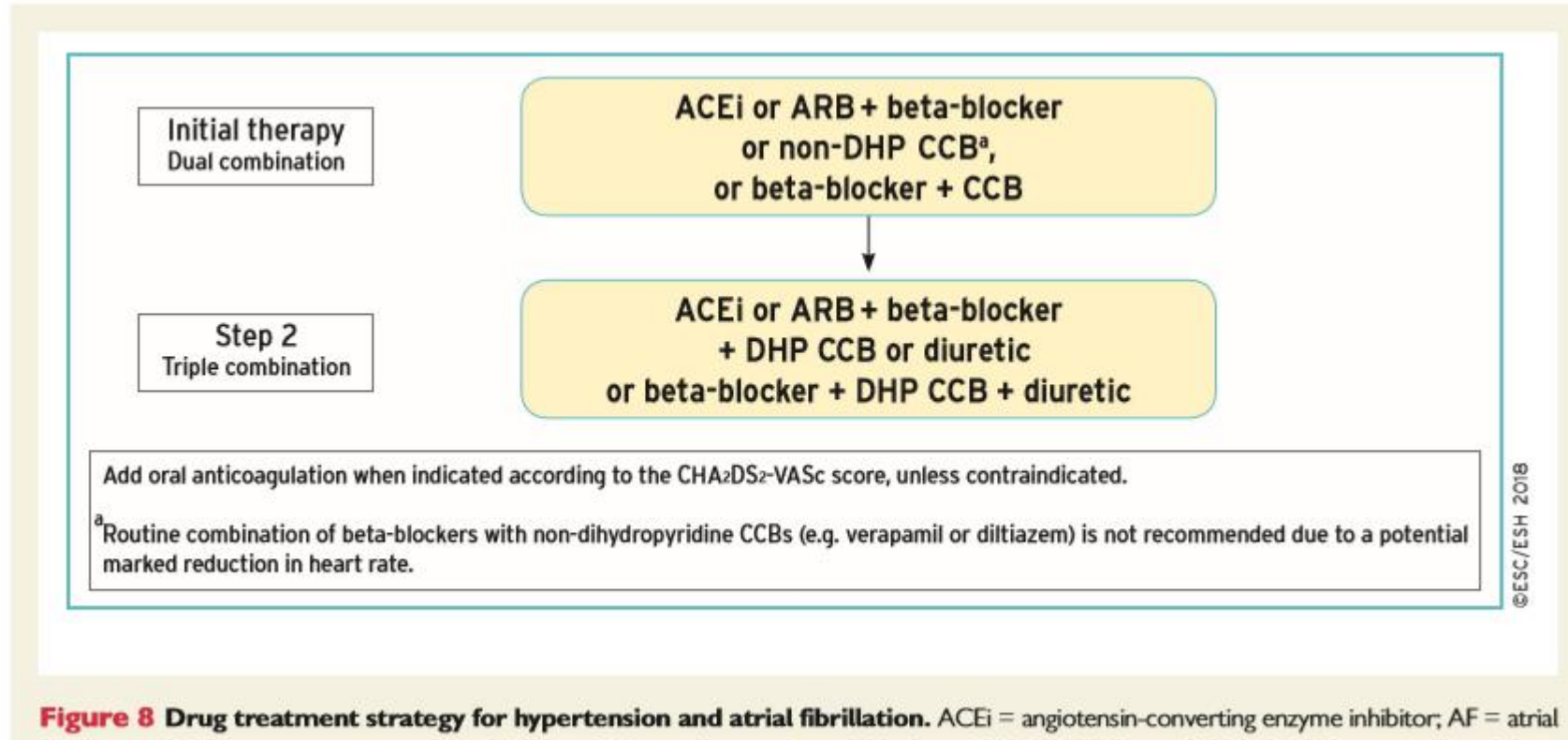


Figure 5 Drug treatment strategy for hypertension and coronary artery disease. ACEi = angiotensin-converting enzyme inhibitor
ARB = angiotensin receptor blocker, BP = blood pressure, CCB = calcium channel blocker, CVD = cardiovascular disease, o.d. = once daily

Kalp yetersizliği ve Hipertansiyon ..



Tek tablette 3'lü kombinasyon? Bazen ilaç uyumu için iyi bazen hipotansif?



İlaç yan etkilerini anlatıyor muyuz?

- ACE-i öksürük
- Amlodipin ayakta ödem
- Diüretiklerin yan etkilerini
 - Hiponatremi-huzursuz, ajite, uykusuz, yorgun, halsizlik
 - Hipopotasemi-yorgun, halsiz, kas güçsüzlüğü, solunum güçsüzlüğü, KABZILIK, aritmiler
 - Hiperürisemi (ürik asit yüksekliği ev GUT belirtileri)

Antihipertansif ilaçların yan etkileri hastalara anlatılmalı ve sorgulanmalı

- **Cardura ortostatik hipotansiyon**
- **Beta blokerler adinami ve erektil disfonksiyon**
- **Verapamil veya diltizem kabızlık**
- **Aldacton ağırlı jinekomasti**

Dirençli hipertansiyon(Tolere edilebilen en yüksek doz Ace-i veya ARB + KKB +diüretik (indapamid veya klortalidon)

- Psödodirençli HT: Hasta ilaçlarını düzenli almıyor. Sonrada düşmüyor diye geliyor...

• Uyku-apnea sendromu...

- Psödoefedrin (enerji içecekleri, otrivin, iliadin, tylol hot, neurofen cold, aferin vb)alıyor
- Efexor veya cymbalta alıyor
- Akut koroner iskeminin veya TİA ilk belirtisi*****
- Psödofeokromositoma (yaygın anksiyete bozukluğu)

Dirençli sekonder hipertansiyonda önemli bir sebep; Primer hipertaldosteronizm

- Sürrenalde hiperplazi (Surrenal BT ya da MR)
- **Kan tetkiki ile**
 - **PRA(plazma renin aktivitesinde azalma)**
 - **PAK (plazma aldosteron konsantrasyonunda artma)**
 - **ARR(aldosteron renin oranında) artma**
- TEDAVİ; spironolakton 100 mg 1x1 gibi

Psödofeokromositoma (yaygın anksiyete bozukluğu)

- Diğer sekonder hipertansiyon sebepleri (psödoefedrin veya AKS vb) ekarte edildikten sonra hemen feo vb için MR/metanefrin istemeden önce hastada anksiyete yapıcı bir sebep??
- Organik bir sebep yoksa panik atak hipertansiyonu için en iyi tedavi, yoksa her gün size tansiyonun yüksek diye gelir.
- ***Xanax 0.5 mg 2x1/2 (3 hafta) + Dideral 3x1/2 (3-6 ay) + Cipralex 10 mg 1x1 (6-12) ay***

Gebelikte hipertansiyon

- Gebelikte öncesinde varsa veya subklinik organ hasarı veya semptomu varsa 140/90 mmHg üstü ilaç öneriliyor
- *Metildopa, labetalol ve gerekirse KKB (amlodipin, nifedipin) öneriliyor
- **ACE-i ve ARB kontrendike
- *Yukarıdakiler yoksa gebelikte 150 /95 üstü öneriliyor
- *Gebelikte 170/110 üstü acil yatış

Aman Dikkat

- **Üreme çağındaki kadınlarda çok özel proteinüri veya kalp yetersizliği vb yoksa (izole HT ise) ilk trcih ACE-i veya ARB olmamalı.**

Gebede ve lohusada hipertansiyon

8.9.1.6 Hypertension and breastfeeding

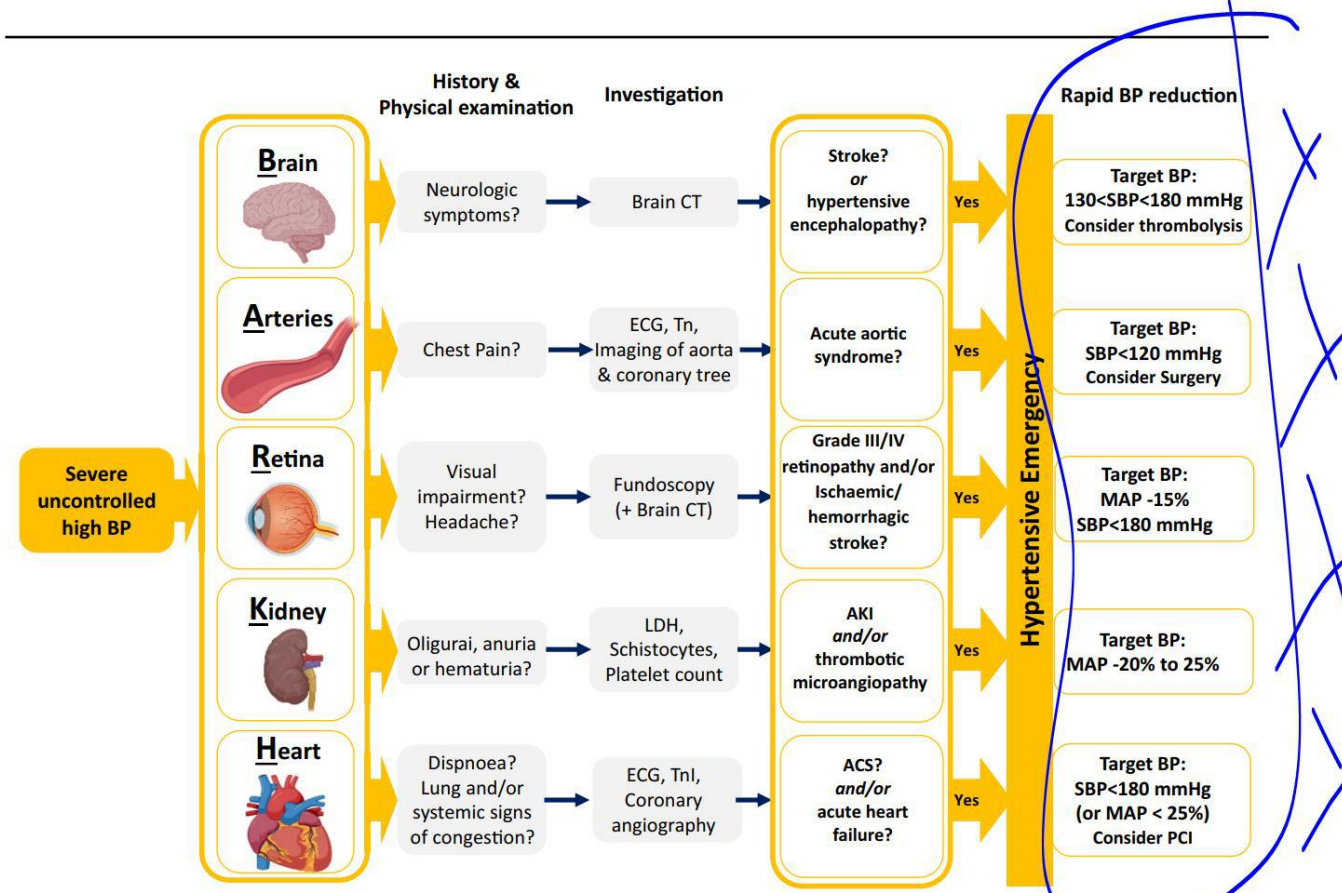
All antihypertensive drugs taken by the nursing mother are excreted into breast milk. Most are present at very low concentrations except for propranolol and nifedipine, with breast milk concentrations similar to those in maternal plasma. Reference to prescribing information in breastfeeding women is important.

Blood pressure post-partum. Post-partum hypertension is common in the first week. Any drug recommended can be used according to the hypertension treatment algorithm shown in Figure 4, with the caveats: (i) methyldopa should be avoided because of the risk of post-partum depression and (ii) consideration should be given to drug choice in breastfeeding women.

Hipertansif acil dışı yüksek tansiyon atakları

- **Kapril çiğneme ve dil altına getirme; etkisi 30 dk başlar ve 2 saatte pik yapar ve 4-6 saat sürer, peş peşe düşmedi diye 30 dk ara ile 3-4 tane kapril alıp ciddi hipotansiyona giren hasta var**
- **Kapril verilmezse cardura 4 mg oral (ilk doz senkopa dikkat)**
- **Kapril vb 1-2 saat sonra (hipertansif acil değilse) eve gidebilir, bu durum son günlerde sık oluyor der ise oral amlidipin vb ekleyerek (kaprilden 6 saat sonra ilk dozu al diyerek) kardiologa gitmesini önererek gönderebiliriz**
- **Ek psödoefedrin vb sormal**

Özellikle hipertansif acillerde ani düşülerden kaçınmalı-Kaş yapayım derken göz çıkarmak



Hipertansif acil ise sevk edilmeli

Fig.2 The BARKH approach is a simplified symptom-based hierarchical algorithm that can assist physicians in the management of the patients presenting with suspected HEs and in the treatment if HE is confirmed. See text for the details. *BARKH* brain, arteries,

retina, kidney and/or heart, *BP* blood pressure, *HE* hypertensive emergency, *LDH* lactic dehydrogenase, *MAP* mean arterial pressure, *SBP* systolic BP

Uyku-apne ve Kardiyovasküler hastalıklar

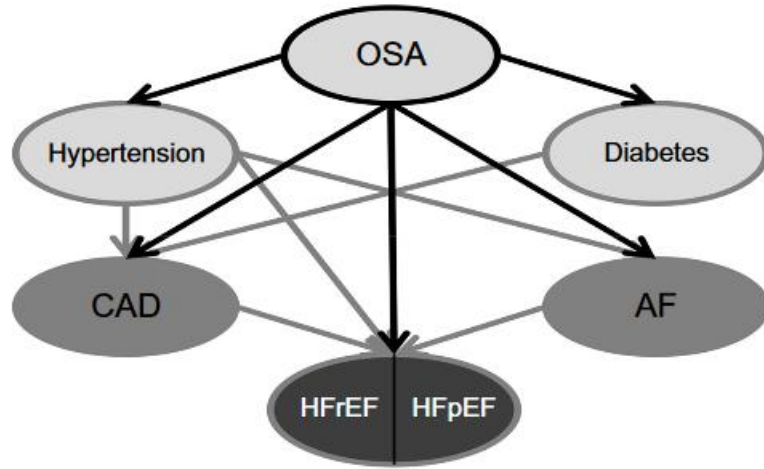


Figure 2 Illustration of the possible interplay between classical cardiovascular risk factors and cardiac diseases, and the role of OSA.

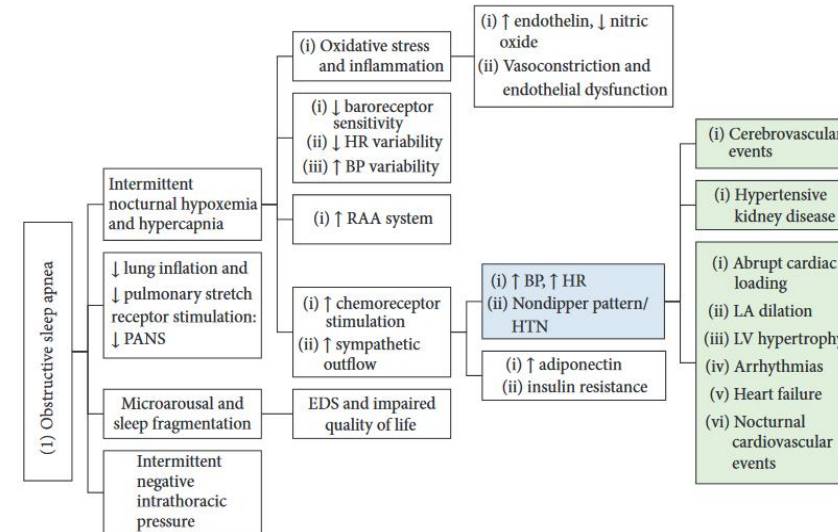


FIGURE 1: Mechanism of hypertension and end organ damage in obstructive sleep apnea. PANS, parasympathetic autonomic nervous system; RAA, renin-angiotensin-aldosterone; EDS, excessive daytime sleepiness; LV, left ventricular; LA, left atrial.



BMC Res Notes. 2012 Oct 29;5:591. doi: 10.1186/1756-0500-5-591.

Hypertension in a young boy: an energy drink effect.

Usman A¹, Jawaid A.

⊕ Author information

Abstract

BACKGROUND: Use of energy drinks has significantly increased in recent times. Besides athletes, teenagers and students are among the most common consumers. However, popularity is also increasing among the younger and older age groups. Most of the users believe that they are a good source of instant energy and are unaware of its high Caffeine content resulting in harmful effects on health.

CASE PRESENTATION: We report the case of a young boy who presented with palpitations and high blood pressure as a result of energy drinks usage. He had been consuming a "Sting" energy drink on regular basis while studying for long hours during his O' level Exams. His medical examination revealed Sinus tachycardia and high blood pressure. Rest of the examination and lab workup was within normal limits. His pulse and blood pressure returned to normal range after discontinuing Sting usage.

CONCLUSION: Several studies have reported numerous health hazards including cardiac effects associated with energy drinks. Warning labeling should be done on these drinks regulating the content of Caffeine and its harmful effects on health.

PMID: 23106965 PMCID: PMC3507669 DOI: 10.1186/1756-0500-5-591

[Indexed for MEDLINE] [Free PMC Article](#)



Treatment of cocaine cardiovascular toxicity: a systematic review.

Richards JR¹, Garber D¹, Laurin EG¹, Albertson TE², Derlet RW¹, Amsterdam EA³, Olson KR⁴, Ramoska EA⁵, Lange RA⁶.

⊕ Author information

Abstract

INTRODUCTION: Cocaine abuse is a major worldwide health problem. Patients with acute cocaine toxicity presenting to the emergency department may require urgent treatment for tachycardia, dysrhythmia, hypertension, and coronary vasospasm, leading to pathological sequelae such as acute coronary syndrome, stroke, and death.

OBJECTIVE: The objective of this study is to review the current evidence for pharmacological treatment of cardiovascular toxicity resulting from cocaine abuse.

METHODS: MEDLINE, PsycINFO, Database of Abstracts of Reviews of Effects (DARE), OpenGrey, Google Scholar, and the Cochrane Library were searched from inception to November 2015. Articles on pharmacological treatment involving human subjects and cocaine were selected and reviewed. Evidence was graded using Oxford Centre for Evidence-Based Medicine guidelines. Treatment recommendations were compared to current American College of Cardiology/American Heart Association guidelines. Special attention was given to adverse drug events or treatment failure. The search resulted in 2376 articles with 120 eligible involving 2358 human subjects. Benzodiazepines and other GABA-active agents: There were five high-quality (CEBM Level I/II) studies, three retrospective (Level III), and 25 case series/reports (Level IV/V) supporting the use of benzodiazepines and other GABA-active agents in 234 subjects with eight treatment failures. Benzodiazepines may not always effectively mitigate tachycardia, hypertension, and vasospasm from cocaine toxicity. Calcium channel blockers: There were seven Level I/II, one Level III, and seven Level IV/V studies involving 107 subjects and one treatment failure. Calcium channel blockers may decrease hypertension and coronary vasospasm, but not necessarily tachycardia. Nitric oxide-mediated vasodilators: There were six Level I/II, one Level III, and 25 Level IV/V studies conducted in 246 subjects with 11 treatment failures and two adverse drug events. Nitroglycerin may lead to severe hypotension and reflex tachycardia. Alpha-adrenoceptor blocking drugs: There were two Level I studies and three case reports. Alpha-1 blockers may improve hypertension and vasospasm, but not tachycardia, although evidence is limited. Alpha-2-adrenoceptor agonists: There were two high-quality studies and one case report detailing the successful use of dexmedetomidine. Beta-blockers and β/α -blockers: There were nine Level I/II, seven Level III, and 34 Level IV/V studies of β -blockers, with 1744 subjects, seven adverse drug events, and three treatment failures. No adverse events were reported for use of combined β/α blockers.

Psödoefedrin ve ölüm...kokain ve ölüm...redbull ve ölüm...

Pseudoephedrine

Adverse effects

- CNS stimulation- insomnia, nervousness, excitability, dizziness and anxiety.
- Tachycardia
- Arrhythmias
- Hypertension
- Hallucinations
- Seizures
- Systemic contact dermatitis
- Paranoid psychosis
- Stroke



Hiperlipidemi tedavisi

ESC 2019-dislipidemi kılavuzu

Sorunlar

- Primer korumayı bırakalım sekonder koruma (bilinen stent/bypass/Mi/inme/periferik arter hasta vb aterosklerotik KV hastalığı olan hastaların neredeyse yarısı yazılan statini almıyor.
- Statinler karaciğere dokunuyor, demans, unutkanlık veya cinsel disfonksiyon yapıyor diye kesen kesene....
- **Öncelikle statin acil ve mutlaka ve ömür boyu alması gereken bu sekonder KV hastalığı olan gruba**
 - 1-Statin kullanımının önemi anlatılmalı,
 - 2-Yan etki az denilmeli,
 - 3-Kolesterolün ne kadar baş belası olduğu ve damarı tıkadığı anlatılmalı,
 - 4-100 derece fayda verip 2 derece zarar verdiği anlatılmalı ve pleotropik etkilerinden (bir taşla 5 kuş vurduğumuz) bahsedilmeli

Sorunlar

- Statin alıyor ve 3 ay sonra kolesterol normale gelince kesiyor...Ve tekrar yükseleceğini bilmiyor.
- **Kolesterolümüzün %70-80'ı endojen yapılı, vejeteryanda olsanız ailesel geçiş varsa yine hiperkolesterolemi olabilirsiniz**
- Kas ağrısı yapıyor diye kesilen var
- **Statin düşük doz verilip ilacı alıyor duygusuna kapılmak. Hedef değerleri önemsememek. Stent ya da bypass var, 10 mg atorvastatin alıyor ve LDL hala 130 ve üstü (ideali 5 mg/dl altı). MAKSİMUM DOZA ÇIKMA HEYECANI YOK**

İlk tercih statinler olmalı

Pharmacological LDL-C lowering

If the goals are not achieved with the maximum tolerated dose of statin, combination with ezetimibe is recommended.

Pharmacological LDL-C lowering

For secondary prevention, patients at very-high risk not achieving their goal on a maximum tolerated dose of statin and ezetimibe, a combination with a PCSK9 inhibitor is recommended.

For very-high-risk FH patients (that is, with ASCVD or with another major risk factor) who do not achieve their goals on a maximum tolerated dose of statin and ezetimibe, a combination with a PCSK9 inhibitor is recommended.

Continued

- Hiperlipidemi tedavisinin Messi ya da Ronaldosu **STATİNLERDİR. Gerekğinde maksimum doza çıkılmalı**
- Statin maksimum doza rağmen yetmez ise ek olarak ezetimib eklenebilir
- Statin ve ezetimibe rağmen yine hedef kolesterol değerlerine ulaşamazsa **PCSK9 inhibitörleri (proprotein convertase subtilisin/kexin type 9 inhibitörleri; alirocumab ve evolocumab vb) eklenebilir**

Özellikle sekonder koruma grubunda LDL 55 mg/dl üstü ve primer korumanın çok yüksek risk grubuna LDL 70 ve üstü mutlaka statin verilmeli

- Akut Mi döneminde LDL bakılmaksızın hemen hastaya statin yüksek doz verilmeli (amaç pleotropik etki)
- **Pleotropik etki (ilaç ana etkisi dışında ek diğer olumlu etkileri)**
- **Statinlerin pleotropik etkisi**
 - **Antitrombotik**
 - **Antiinflamatuvar (CRP düşürücü)**
 - **Antiendotelial disfonksiyon düzeltici**

Hedef kolesterol düzeyi ve ilaç başlama endikasyonunu belirleyen 10 yılda beklenen KV bir olay (Mi, inme vb) gelişme riski hesaplanmalı

Table 4 Cardiovascular risk categories

Very-high-risk	People with any of the following: Documented ASCVD, either clinical or unequivocal on imaging. Documented ASCVD includes previous ACS (MI or unstable angina), stable angina, coronary revascularization (PCI, CABG, and other arterial revascularization procedures), stroke and TIA, and peripheral arterial disease. Unequivocally documented ASCVD on imaging includes those findings that are known to be predictive of clinical events, such as significant plaque on coronary angiography or CT scan (multivessel coronary disease with two major epicardial arteries having >50% stenosis), or on carotid ultrasound. DM with target organ damage, ^a or at least three major risk factors, or early onset of T1DM of long duration (>20 years). Severe CKD (eGFR <30 mL/min/1.73 m ²). A calculated SCORE ≥10% for 10-year risk of fatal CVD. FH with ASCVD or with another major risk factor.
High-risk	People with: Markedly elevated single risk factors, in particular TC >8 mmol/L (>310 mg/dL), LDL-C >4.9 mmol/L (>190 mg/dL), or BP ≥180/110 mmHg. Patients with FH without other major risk factors. Patients with DM without target organ damage, ^a with DM duration ≥10 years or another additional risk factor. Moderate CKD (eGFR 30–59 mL/min/1.73 m ²). A calculated SCORE ≥5% and <10% for 10-year risk of fatal CVD.
Moderate-risk	Young patients (T1DM <35 years; T2DM <50 years) with DM duration <10 years, without other risk factors. Calculated SCORE ≥1 % and <5% for 10-year risk of fatal CVD.
Low-risk	Calculated SCORE <1% for 10-year risk of fatal CVD.

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- **Çok yüksek KV risk grubunda olanlar**
 - Bilinen Mi, stent, bypass, statble angina, PAH, karotis darlık, inme geçirenler
- Ya da kesinleşmiş ve gösterilmiş örneğin koroner anjio ile 1 damarda en az %50 ve üstü darlık veya koroner BT anjioda en az 2 damarda %50 ve üstü darlık veya karotiste anlamlı darlık saptanmışsa
- Ya da retinopati/proteinüri vb organ tutulumu olan DM veya en az 3 majör risk faktörü ile beraber olan DM veya erken başlangıçlı ve 20 yılı aşmış tip 1 DM
- Ya da ciddi böbrek yetersizliği olan hasta (GFR 30'un altı)
- SCORE risk indeksi %10 ve üstü ise

Hedef kolesterol düzeyi ve ilaç başlama endikasyonunu belirleyen 10 yılda beklenen KV bir olay (Mİ, inme vb) gelişme riski hesaplanmalı

Table 4 Cardiovascular risk categories

Very-high-risk	People with any of the following: Documented ASCVD, either clinical or unequivocal on imaging. Documented ASCVD includes previous ACS (MI or unstable angina), stable angina, coronary revascularization (PCI, CABG, and other arterial revascularization procedures), stroke and TIA, and peripheral arterial disease. Unequivocally documented ASCVD on imaging includes those findings that are known to be predictive of clinical events, such as significant plaque on coronary angiography or CT scan (multivessel coronary disease with two major epicardial arteries having >50% stenosis), or on carotid ultrasound. DM with target organ damage, ^a or at least three major risk factors, or early onset of T1DM of long duration (>20 years). Severe CKD (eGFR <30 mL/min/1.73 m ²). A calculated SCORE ≥10% for 10-year risk of fatal CVD. FH with ASCVD or with another major risk factor.
High-risk	People with: Markedly elevated single risk factors, in particular TC >8 mmol/L (>310 mg/dL), LDL-C >4.9 mmol/L (>190 mg/dL), or BP ≥180/110 mmHg. Patients with FH without other major risk factors. Patients with DM without target organ damage, ^a with DM duration ≥10 years or another additional risk factor. Moderate CKD (eGFR 30–59 mL/min/1.73 m ²). A calculated SCORE ≥5% and <10% for 10-year risk of fatal CVD.
Moderate-risk	Young patients (T1DM <35 years; T2DM <50 years) with DM duration <10 years, without other risk factors. Calculated SCORE ≥1 % and <5% for 10-year risk of fatal CVD.
Low-risk	Calculated SCORE <1% for 10-year risk of fatal CVD.

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- Yüksek KV risk grubunda olanlar
 - Tek majör risk faktörü belirgin var ise (TK 310 veya LDL 190 üstü veya TA180/110 mmHG üstü)
 - Tek başına ailesel hiperkolesterolemi varsa
 - DM olup hedef organ tutulumu olmayan ve çok yüksek risk grubu dışında kalanlar
 - GFR 30-59 arası KBY hastaları
 - SCORE risk puanı %5-10 arası olanlar
- Orta KV derecede risk grubunda olanlar
 - 35 yaş altı tip 1 Dm veya 50 yaş altı tip 2 Dm var ama DM süresi 10 yıldan az olan ve ek majör risk faktörü olmayan DM hastalar
 - SCORE risk indeksi %1-5 puan arasında olanlar
- Düşük KV risk grubunda olanlar
 - SCORE risk puanı %1'in altı olanlar

ESC SCORE 2/SCORE OP

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Choose your risk calculator category



Up to 10 year risk calculators



Lifetime risk calculators

Get help for using calculator and application



Find the right calculator



How to use this application

All risk calculators ↑

Total CV risk (SCORE) %	
Primary prevention	<1, low-risk
	Class ^a /Level ^b
	≥1 to <5, or moderate risk (see Table 4)
	Class ^a /Level ^b
	≥5 to <10, or high-risk (see Table 4)
	Class ^a /Level ^b
	≥10, or at very-high risk due to a risk condition (see Table 4)
	Class ^a /Level ^b
Secondary prevention	Very-high-risk

Heart Risk Calculator

Home About Contact

Calculate your 10-year risk of heart disease or stroke using the ASCVD algorithm published in 2013 ACC/AHA Guideline on the Assessment of Cardiovascular Risk.

This calculator assumes that you have not had a prior heart attack or stroke. If you have, generally it is recommended that you discuss with your doctor about starting aspirin and a statin. Furthermore, if you have an LDL-cholesterol (bad cholesterol) greater than 190, it is also generally recommended that you discuss with your doctor about starting aspirin and a statin.

Unfortunately, there is insufficient data to reliably predict risk for those less than 40 years of age or greater than 79 years of age and for those with total cholesterol greater than 320.

UPDATE (11/21/17) – The ACC/AHA has released their 2017 Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults. At a high level, the new guidelines redefine hypertension as blood pressure >130/80 and recommend starting anti-hypertensives based on ASCVD risk score of >10%. This will be a significant change from JNC-8. Please let us know if you would like us to incorporate the new guidelines into cvriskcalculator.com by completing this 1-question survey.

UPDATE (6/30/16) – The calculator has been vetted against the final guidelines from the USPSTF for initiating aspirin therapy.

UPDATE (9/18/15) – The calculator now also incorporates draft guidelines from the USPSTF for

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Calculator: Cardiovascular risk assessment (10-year, men: Patient education)

Input

Age 53 yr
Systolic blood pressure mmHg
Total cholesterol 240 mg/dL
HDL cholesterol 49 mg/dL
On blood pressure medication No (1,93303)
Cigarette smoker No (0)
Diabetes present No (0)

Results

Risk %

Reset form

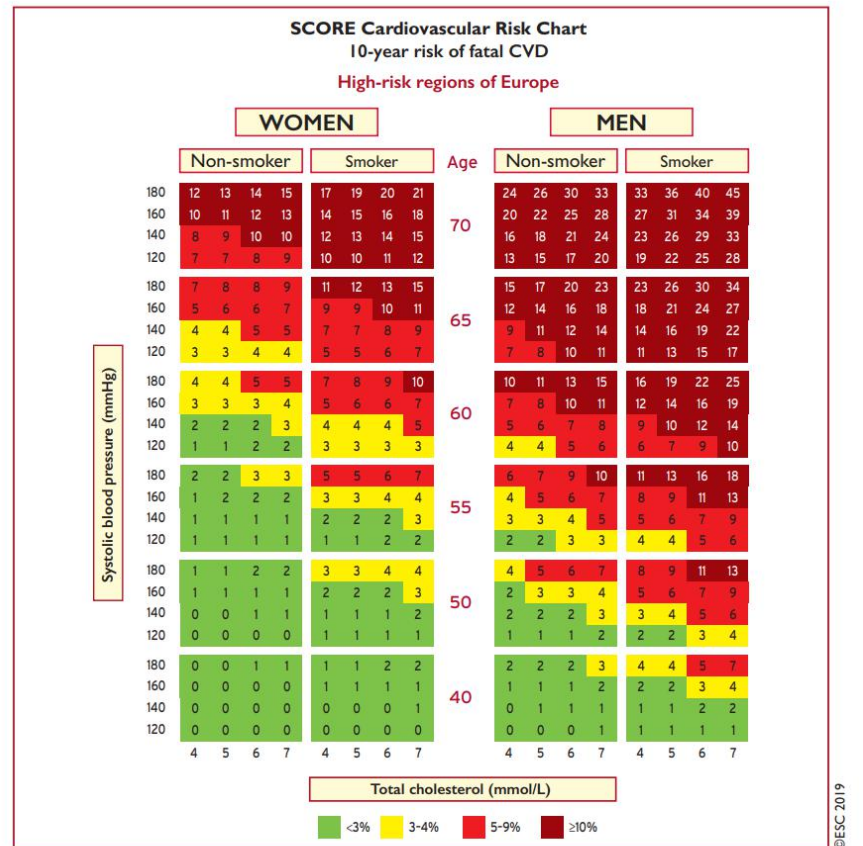


Figure 1 Systematic Coronary Risk Estimation chart for European populations at high cardiovascular disease risk. The 10-year risk of fatal cardiovascular

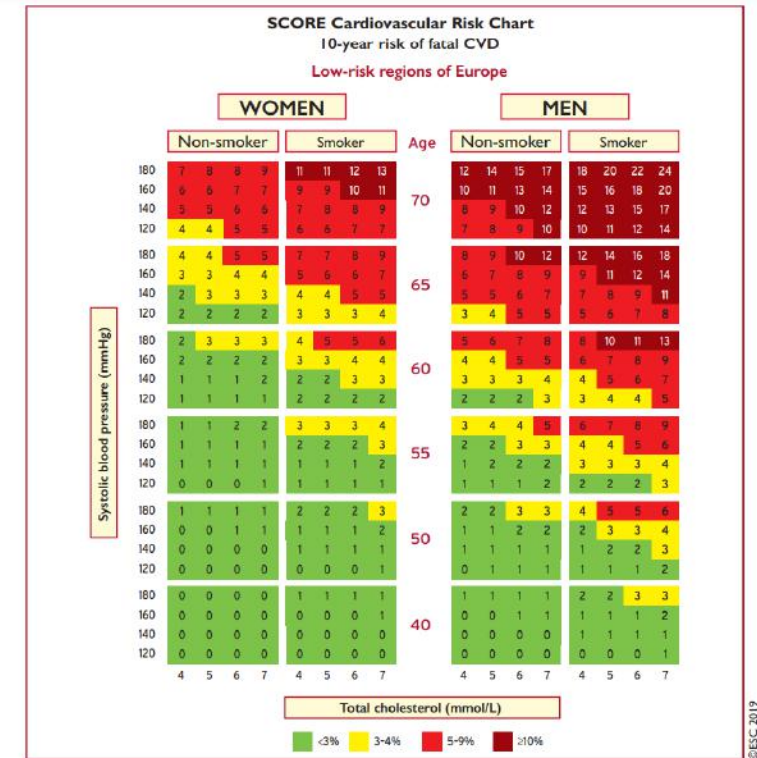


Figure 2 Systematic Coronary Risk Estimation chart for European populations at low cardiovascular disease risk. The 10-year risk of fatal cardiovascular disease in populations at low cardiovascular disease risk based on the following risk factors: age, gender, smoking, systolic blood pressure, and total cholesterol. To convert the risk of fatal cardiovascular disease to risk of total (fatal + non-fatal) cardiovascular disease, multiply by 3 in men and by 4 in women, and slightly less in older people. Note: the Systematic Coronary Risk Estimation chart is for use in people without overt cardiovascular disease, diabetes (type 1 and 2), chronic kidney disease, familial hypercholesterolaemia, or very high levels of individual risk factors because such people are already at high-risk and need intensive risk factor management. Cholesterol: 1 mmol/L=38.67 mg/dL. The SCORE risk charts presented above differ slightly from those in the 2016 European Society of Cardiology/European Atherosclerosis Society Guidelines for the management of dyslipidaemias and the 2016 European

Sekonder koruma grubuna LDL değeri ne olursa olsun hemen statin başla (LDL 55 mg/dl altı olsa da; pleotropik etki)

Table 5 Intervention strategies as a function of total cardiovascular risk and untreated low-density lipoprotein cholesterol levels

		Untreated LDL-C levels					
Total CV risk (SCORE) %		<1.4 mmol/L (55 mg/dL)	1.4 to <1.8 mmol/L (55 to <70 mg/dL)	1.8 to <2.6 mmol/L (70 to <100 mg/dL)	2.6 to <3.0 mmol/L (100 to <116 mg/dL)	3.0 to <4.9 mmol/L (116 to <190 mg/dL)	≥4.9 mmol/L (≥190 mg/dL)
Primary prevention	<1, low-risk	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	I/C	I/C	I/C	I/C	IIa/A	IIa/A
	≥1 to <5, or moderate risk (see Table 4)	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	I/C	I/C	IIa/A	IIa/A	IIa/A	IIa/A
	≥5 to <10, or high-risk (see Table 4)	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	IIa/A	IIa/A	IIa/A	I/A	I/A	I/A
	≥10, or at very-high risk due to a risk condition (see Table 4)	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
Secondary prevention	Class ^a /Level ^b	IIa/B	IIa/A	I/A	I/A	I/A	I/A
	Very-high-risk	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	IIa/A	I/A	I/A	I/A	I/A	I/A

Primer korumanın çok yüksek risk grubunda ise ne zaman statin?

Table 5 Intervention strategies as a function of total cardiovascular risk and untreated low-density lipoprotein cholesterol levels

		Untreated LDL-C levels					
Total CV risk (SCORE) %		<1.4 mmol/L (55 mg/dL)	1.4 to <1.8 mmol/L (55 to <70 mg/dL)	1.8 to <2.6 mmol/L (70 to <100 mg/dL)	2.6 to <3.0 mmol/L (100 to <116 mg/dL)	3.0 to <4.9 mmol/L (116 to <190 mg/dL)	≥4.9 mmol/L (≥190 mg/dL)
Primary prevention	<1, low-risk	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	I/C	I/C	I/C	I/C	IIa/A	IIa/A
	≥1 to <5, or moderate risk (see Table 4)	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	I/C	I/C	IIa/A	IIa/A	IIa/A	IIa/A
	≥5 to <10, or high-risk (see Table 4)	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	IIa/A	IIa/A	IIa/A	I/A	I/A	I/A
Secondary prevention	≥10, or at very-high risk due to a risk condition (see Table 4)	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	IIa/B	IIa/A	I/A	I/A	I/A	I/A
	Very-high-risk	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
Class ^a /Level ^b		IIa/A	I/A	I/A	I/A	I/A	I/A

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- Bazal 55 mg/dl altı olduğu sürece ise ilaç verilmez, sadece YTD (yaşam tarzı değişikliği) önerilir.
- LDL 70 mg/dl üstü hemen ilaç statin başlanır.
- Bazal 55-70 mg arası ise 3 ay ilaç vermeden yaşam tarzı değişikliği şansı verilebilir ve 55'in altına düşerse sadece YTD devam, ama YTD rağmen 55 üstü olursa statin eklenir

Sekonder koruma için (Mİ/STENT vb) veya primer korunma açısından çok yüksek risk grubunda olanlarda

• New/revised concepts
More intensive reduction of LDL-C across CV risk categories
- For secondary prevention in very-high-risk patients, an LDL-C reduction of $\geq 50\%$ from baseline and an LDL-C goal of <1.4 mmol/L (<55 mg/dL) are recommended. - For patients with ASCVD who experience a second vascular event within 2 years (not necessarily of the same type as the first event) while taking maximally tolerated statin therapy, an LDL-C goal of <1.0 mmol/L (<40 mg/dL) may be considered. - In primary prevention, for individuals at very-high risk but without FH, an LDL-C reduction of $\geq 50\%$ from baseline and an LDL-C goal of <1.4 mmol/L (<55 mg/dL) are recommended. For individuals at very-high risk (that is, with another risk factor but without ASCVD), in primary prevention the same goals for LDL-C lowering should be considered. - For patients at high risk, an LDL-C reduction of $\geq 50\%$ from baseline and an LDL-C goal of <1.8 mmol/L (<70 mg/dL) are recommended. - For individuals at moderate risk, an LDL-C goal of <2.6 mmol/L (<100 mg/dL) should be considered. - For individuals at low risk, an LDL-C goal of <3.0 mmol/L (<116 mg/dL) may be considered.
The rationale for the revised, lower LDL-C goals across CV risk categories is discussed, based on a critical synthesis of available evidence from lipid-modifying interventions resulting in reductions in CV risk.
Pharmacological LDL-C-lowering strategies
The section on pharmacological strategies to lower LDL-C emphasizes the concept that the absolute LDL-C reduction (determined by pre-treatment LDL-C levels and the LDL-lowering efficacy of the medications) dictates the relative risk reduction, which in turn—depending on the baseline CV risk—defines the associated absolute CV risk reduction in individual patients.
Risk classification in patients with FH
Patients with FH and ASCVD, or another major risk factor, are classified as very-high-risk, and those without known ASCVD and without other risk factors as high-risk. Recommended treatment goals are defined accordingly.
Adverse effects of statins
The distinction between formal statin myopathy vs. so-called statin-associated muscle symptoms is emphasized, and the discordance in reported frequency of symptoms in RCTs vs. observational studies are critically discussed on the basis of new relevant evidence.

- 1-Statın yüksek doz başla (atorvastatin 40-80 mg 1x1 veya rosuvastatin 20-40 mg 1x1) 2-3 ay sonra hala hedef LDL'ye ulaşılmamışsa statine ezetimib ekle ve 2-3 ay sonra ile LDL kolesterole tekrar bakmalı ve yine ulaşılmazsa 3.ek ilaç olarak PCSK9 inhibitörleri eklenmeli
- 2- Hedef LDL; bazale göre %50 ve üzerinde azaltılması ve 55 mg/dl altına indirilmeli.
- Örnekler
- Vaka-1; Bazal LDL 180 mg/dl ise hedef %50 azaltılması durumunda 90 mg7dl olacağından daha da düşürmek ve 55 mg/dl olması hedeflenmeli
- Vaka-2;Bazal 80 mg/dl ise hedef 55 mg/dl altı değil bu kez %50 azaltmak olmalı yani 40 mg/Dl altına düşürülmeli
- Kısacası bazal LDL 110 mg/dl ve üstü ise 55 mg/dl altı, bazal 110 altı ise %50 kuralı geçerli olmalı

Yüksek risk grubunda olanlarda hedef LDL ne olmalı?

Bu grup sekonder koruma değil primer koruma grubudur. Sekonder koruma otomatik olarak çok yüksek gruba girer

• New/revised concepts
More intensive reduction of LDL-C across CV risk categories
- For secondary prevention in very-high-risk patients, an LDL-C reduction of $\geq 50\%$ from baseline and an LDL-C goal of <1.4 mmol/L (<55 mg/dL) are recommended. - For patients with ASCVD who experience a second vascular event within 2 years (not necessarily of the same type as the first event) while taking maximally tolerated statin therapy, an LDL-C goal of <1.0 mmol/L (<40 mg/dL) may be considered. - In primary prevention, for individuals at very-high risk but without FH, an LDL-C reduction of $\geq 50\%$ from baseline and an LDL-C goal of <1.4 mmol/L (<55 mg/dL) are recommended. For individuals at very-high risk (that is, with another risk factor but without ASCVD), in primary prevention the same goals for LDL-C lowering should be considered. - For patients at high risk, an LDL-C reduction of $\geq 50\%$ from baseline and an LDL-C goal of <1.8 mmol/L (<70 mg/dL) are recommended. - For individuals at moderate risk, an LDL-C goal of <2.6 mmol/L (<100 mg/dL) should be considered. - For individuals at low risk, an LDL-C goal of <3.0 mmol/L (<116 mg/dL) may be considered. The rationale for the revised, lower LDL-C goals across CV risk categories is discussed, based on a critical synthesis of available evidence from lipid-modifying interventions resulting in reductions in CV risk.
Pharmacological LDL-C-lowering strategies
The section on pharmacological strategies to lower LDL-C emphasizes the concept that the absolute LDL-C reduction (determined by pre-treatment LDL-C levels and the LDL-lowering efficacy of the medications) dictates the relative risk reduction, which in turn—depending on the baseline CV risk—defines the associated absolute CV risk reduction in individual patients.
Risk classification in patients with FH
Patients with FH and ASCVD, or another major risk factor, are classified as very-high-risk, and those without known ASCVD and without other risk factors as high-risk. Recommended treatment goals are defined accordingly.
Adverse effects of statins
The distinction between formal statin myopathy vs. so-called statin-associated muscle symptoms is emphasized, and the discordance in reported frequency of symptoms in RCTs vs. observational studies are critically discussed on the basis of new relevant evidence.

- Bazal 70 mg/dl altı ise ilaç verilmez, sadece YTD (yaşam tarzı değişikliği) önerilir.
- LDL 100 mg/dl üstü hemen ilaç statin başlanır.**
- Bazal 70-100 mg arası ise 3 ay ilaç vermeden yaşam tarzı değişikliği şansı verilebilir ve 70'in altına düşerse sadece YTD devam, ama YTD rağmen 70 üstü olursa statin eklenir**
- İlaç başlanırsa da hedef LDL; Yüksek risk grubunda olan hastalarda ise LDL tedavi ile bazale göre %50 azaltılmalı ve LDL 70 mg/dl altı olmalı
 - Bazal LDL 140 mg/dl ve üstü ise 70 mg/dl altı kuralı**
 - Bazal 140 altı ise %50 kuralı geçerli olmalı (mesela bazal 100 olsun, tedavi ile hedef 50 mg/dl altı olmalı)

Orta ve düşük risk grubunda olanlarda hedef LDL ne olmalı?

New/revised concepts
More intensive reduction of LDL-C across CV risk categories
- For secondary prevention in very-high-risk patients, an LDL-C reduction of $\geq 50\%$ from baseline and an LDL-C goal of < 1.4 mmol/L (< 55 mg/dL) are recommended. - For patients with ASCVD who experience a second vascular event within 2 years (not necessarily of the same type as the first event) while taking maximally tolerated statin therapy, an LDL-C goal of < 1.0 mmol/L (< 40 mg/dL) may be considered. - In primary prevention, for individuals at very-high risk but without FH, an LDL-C reduction of $\geq 50\%$ from baseline and an LDL-C goal of < 1.4 mmol/L (< 55 mg/dL) are recommended. For individuals at very-high risk (that is, with another risk factor but without ASCVD), in primary prevention the same goals for LDL-C lowering should be considered. - For patients at high risk, an LDL-C reduction of $\geq 50\%$ from baseline and an LDL-C goal of < 1.8 mmol/L (< 70 mg/dL) are recommended. - For individuals at moderate risk, an LDL-C goal of < 2.6 mmol/L (< 100 mg/dL) should be considered. - For individuals at low risk, an LDL-C goal of < 3.0 mmol/L (< 116 mg/dL) may be considered. The rationale for the revised, lower LDL-C goals across CV risk categories is discussed, based on a critical synthesis of available evidence from lipid-modifying interventions resulting in reductions in CV risk.
Pharmacological LDL-C-lowering strategies
The section on pharmacological strategies to lower LDL-C emphasizes the concept that the absolute LDL-C reduction (determined by pre-treatment LDL-C levels and the LDL-lowering efficacy of the medications) dictates the relative risk reduction, which in turn—depending on the baseline CV risk—defines the associated absolute CV risk reduction in individual patients.
Risk classification in patients with FH
Patients with FH and ASCVD, or another major risk factor, are classified as very-high-risk, and those without known ASCVD and without other risk factors as high-risk. Recommended treatment goals are defined accordingly.
Adverse effects of statins
The distinction between formal statin myopathy vs. so-called statin-associated muscle symptoms is emphasized, and the discordance in reported frequency of symptoms in RCTs vs. observational studies are critically discussed on the basis of new relevant evidence.

- Orta risk grubunda LDL 100 mg/dl'nin altında, düşük risk grubunda ise LDL 116 mg/dl'nin altında ise ilaç başlanmaz sadece YTD devam edilir.
- Orta ve düşük risk grubuna LDL 190 mg/dl üstü ise ilk tanıda ilaç statin başlanır.
- Düşük risk grubunda 116-190 arası ise YTD 3 ay verilir ve sonrasında hala 116 üstü ise ilaç başlanması, 116 altı ise YTD tek başına yeterli kabul edilir.
- Orta riskli grupta 100-116 arası LDL varsa önce 3 ay YTD şansı vermek, sonra hala 100 ve üstü ise ilaç başlanması, 100'ün altı ise sadece YTD ile devam edilmesi öneriliyor.
- Orta riskli gruplarda statin başlanırsa hedef LDL tedavi sonrası 100 mg/dl altı, düşük riskli grupta ise hedef LDL 116 mg/dl altına düşürmek olmalı

Bu tabloyu duvara asmalıyız

Table 5 Intervention strategies as a function of total cardiovascular risk and untreated low-density lipoprotein cholesterol levels

	Total CV risk (SCORE) %	Untreated LDL-C levels					
		<1.4 mmol/L (55 mg/dL)	1.4 to <1.8 mmol/L (55 to <70 mg/dL)	1.8 to <2.6 mmol/L (70 to <100 mg/dL)	2.6 to <3.0 mmol/L (100 to <116 mg/dL)	3.0 to <4.9 mmol/L (116 to <190 mg/dL)	≥4.9 mmol/L (≥190 mg/dL)
Primary prevention	<1, low-risk	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	I/C	I/C	I/C	I/C	IIa/A	IIa/A
	≥1 to <5, or moderate risk (see Table 4)	Lifestyle advice	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	I/C	I/C	IIa/A	IIa/A	IIa/A	IIa/A
	≥5 to <10, or high-risk (see Table 4)	Lifestyle advice	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	IIa/A	IIa/A	IIa/A	I/A	I/A	I/A
Secondary prevention	≥10, or at very-high risk due to a risk condition (see Table 4)	Lifestyle advice	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	IIa/B	IIa/A	I/A	I/A	I/A	I/A
	Very-high-risk	Lifestyle intervention, consider adding drug if uncontrolled	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention	Lifestyle intervention and concomitant drug intervention
	Class ^a /Level ^b	IIa/A	I/A	I/A	I/A	I/A	I/A

Trigliserid yüksekliklerinde ne yapmalıyız?

Recommendations for drug treatment of patients with hypertriglyceridaemia

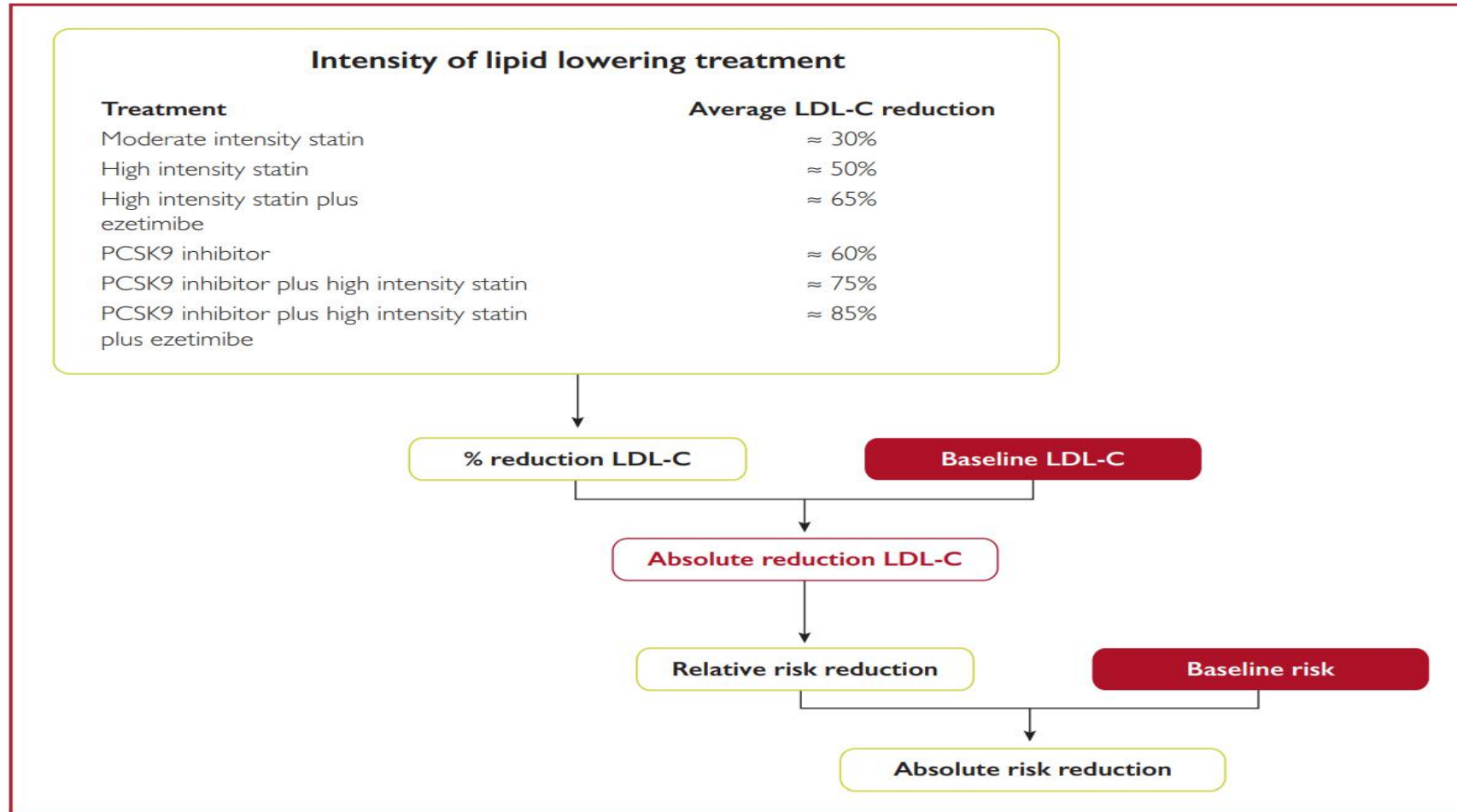
Recommendations	Class ^a	Level ^b
Statin treatment is recommended as the first drug of choice to reduce CVD risk in high-risk individuals with hypertriglyceridaemia [TG levels >2.3 mmol/L (>200 mg/dL)]. ³⁵⁵	I	B
In high-risk (or above) patients with TG levels between 1.5–5.6 mmol/L (135–499 mg/dL) despite statin treatment, n-3 PUFAs (icosapent ethyl 2×2 g/day) should be considered in combination with a statin. ¹⁹⁴	IIa	B
In primary prevention patients who are at LDL-C goal with TG levels >2.3 mmol/L (>200 mg/dL), fenofibrate or bezafibrate may be considered in combination with statins. ^{305–307,356}	IIb	B
In high-risk patients who are at LDL-C goal with TG levels >2.3 mmol/L (>200 mg/dL), fenofibrate or bezafibrate may be considered in combination with statins. ^{305–307,356}	IIb	C

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CVD = cardiovascular disease; LDL-C = low-density lipoprotein cholesterol; PUFA = polyunsaturated fatty acids; TG = triglyceride.

- İdeali 150 mg/dl altı
- **YÜKSEK RİSK GRUBUNDA** olan ve trigliseridi 200 mg/dl üstünde yine tedavi statin
- Statin ve diyetle rağmen düşmezse statine ek olarak fibratlar verilebilir
- **Diyet, spor ve kötü regüle diyabetin tedavisi trigliseridi düşürmek için en iyi yöntem**

LDL-K'ü statin sadece %30, statin + ezetimib +PCSK9 inh 3'lü kullanımında %85 düşürür



Yaşlılarda statin tedavisi

Treatment of dyslipidaemias in older people	
Treatment with statins is recommended for primary prevention, according to the level of risk, in older people aged ≤ 75 .	
Treatment of dyslipidaemias in older people	
Initiation of statin treatment for primary prevention in older people aged >75 may be considered, if at high risk or above.	126

- 75 yaş altı olanlarda statin tedavisi yukarıdaki öneriler geçerlidir
- **75 yaş üstü hastalarda ise**
 - Sekonder korumada statin tedavisi yine yaşdan bağımsız kullanılmalı
 - Ama primer korumada yüksek ve çok yüksek risk grubunda olan hastalarda **klas 2 b** olarak statin tedavisi önerilir

Statin tedavisinin yan etkileri

- En sık (%10-20 hastada) kas ağrısı (myopati-myozit) yapabilir. Çok azında CK yükselir. Çoğunda CK yükselmeden kas ağrısı yapar.
- 1/100.000 rabdomyoliz yapar (CK x10-40 kat ve üstü artması)
- Statin başlanan hastalarda 1.ay ve mümkünse 6 ay ve sonra da yılda bir ALT, AST ve CK bakmalı.
- CK x10 kata kadar statin kesilmez. Ama ister CK yükselsin isterse yükselmeden kas ağrısı yapsın bu durumlarda statin değiştirilip başka statine geçilebilir veya primer korumada ve çok yüksek LDL yoksa gün aşırı statin tedavisi verilebilir ve hastaya mümkünse **kalp için katlanması söylenmeli**

Statin yan etkileri

- **Karaciğer enzimlerinde hafif yükselme %2 hastada görülebilir ki aslında bunların bir kısmı statine bağlı değil hepatosteatoza bağlı**
- **X3 kat KC enzim yükselene kadar statin kesilmez. Statin tedavisi öncesi ALT-AST bakmalı ve bazal değerler bilinmeli**
- **Diabeti kolaylaştırabilir. Aslında çok net değil. Obez hastalarda daha sık statin DM arttırıyor ilişkisi var ki bu durumda net değil ve arttırsada statinin getirdiği fayda daha çok.**

Pravastatin, rosuvastatin ve pitavastatin dışındaki statinlerin aşağıdaki ilaçlarla etkileşimi fazla

Table 10 Drugs potentially interacting with statins metabolized by cytochrome P450 3A4 leading to increased risk of myopathy and rhabdomyolysis

Anti-infective agents	Calcium antagonists	Other
Itraconazole	Verapamil	Ciclosporin
Ketoconazole	Diltiazem	Danazol
Posaconazole	Amlodipine	Amiodarone
Erythromycin		Ranolazine
Clarithromycin		Grapefruit juice
Telithromycin		Nefazodone
HIV protease inhibitors		Gemfibrozil

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Adapted from Egan and Colman,²⁵⁷ and Wiklund et al.²⁵⁸

HIV = human immunodeficiency virus.

TEŞEKKÜRLER