

26. TÜRK KLİNİK MİKROBİYOLOJİ VE İNFEKSİYON HASTALIKLARI KONGRESİ

Hepatit Yönetiminde Metabolik Sağlık

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İnfeksiyon Hastalıkları ve Klinik Mikrobiyoloji Anabilim Dalı

Uydu/Abdi İbrahim

1.5.2026

Neler Konuřulacak?

- ❖ Kronik hepatit B
 - ❖ Tedavisi
- ❖ HBV lipid metabolizma iliřkisi
- ❖ HBV metabolik durum
- ❖ alıřmalarda antiviraller
- ❖ Soru & Yanıt





Global Viral Hepatit Verileri

Table 2.1. Progress towards global viral hepatitis targets, 2022

Indicator	Baseline - 2020	Progress - 2022	Targets - 2025	Targets - 2030
Impact				
Number of new hepatitis B infections per year	1.5 million (1.11 million- 2.09 million)	1.23 million (0.81 million- 1.53 million)	0.85 million (0.62 million- 1.19 million)	170 000 (120 000- 240 000)
	20 per 100 000	16 per 100 000	11 per 100 000	2 per 100 000

Table 2.1. Progress towards global viral hepatitis targets, 2022 (continued)

Indicator	Baseline - 2020	Progress - 2022	Targets - 2025	Targets - 2030
Coverage				
Hepatitis B: percentage of people living with chronic hepatitis B diagnosed	10% against a target of 30%	13.4%	60%	90%
Percentage of people living with chronic hepatitis B treated	2% against target of 30%	2.6%	50%	80%

Tedavi Edin

Tüm yetişkinler ve ergenler (≥ 12 yaş)

World Health Organization 2024

TREAT ALL ADULTS and ADOLESCENTS (aged ≥ 12 years^c)
(including pregnant and non-pregnant women and girls of reproductive age)

1. **SIGNIFICANT FIBROSIS ($\geq F2$) or CIRRHOsis (F4)** (regardless of HBV DNA or ALT levels)

- Clinical criteria for cirrhosis^d
- Non-invasive tests: **APRI >0.5 or transient elastography >7 kPa (adults)^e**

World Health Organization 2024

OR

2. **HBV DNA >2000 IU/mL AND ALT level $> ULN$ ^e**

OR

3. **PRESENCE OF any of following** (regardless of APRI score, HBV DNA or ALT level)

- Coinfection (eg. HIV, HDV, HCV)
- Family history of liver cancer or cirrhosis
- Immune suppression
- **Comorbidities** (eg. diabetes, metabolic dysfunction-associated steatotic liver disease)
- Extrahepatic manifestations (eg. glomerulonephritis or vasculitis)

Antiviral tedaviye başlayın

• **TDF, TAF veya ETV**

• **Osteoporoz, böbrek**

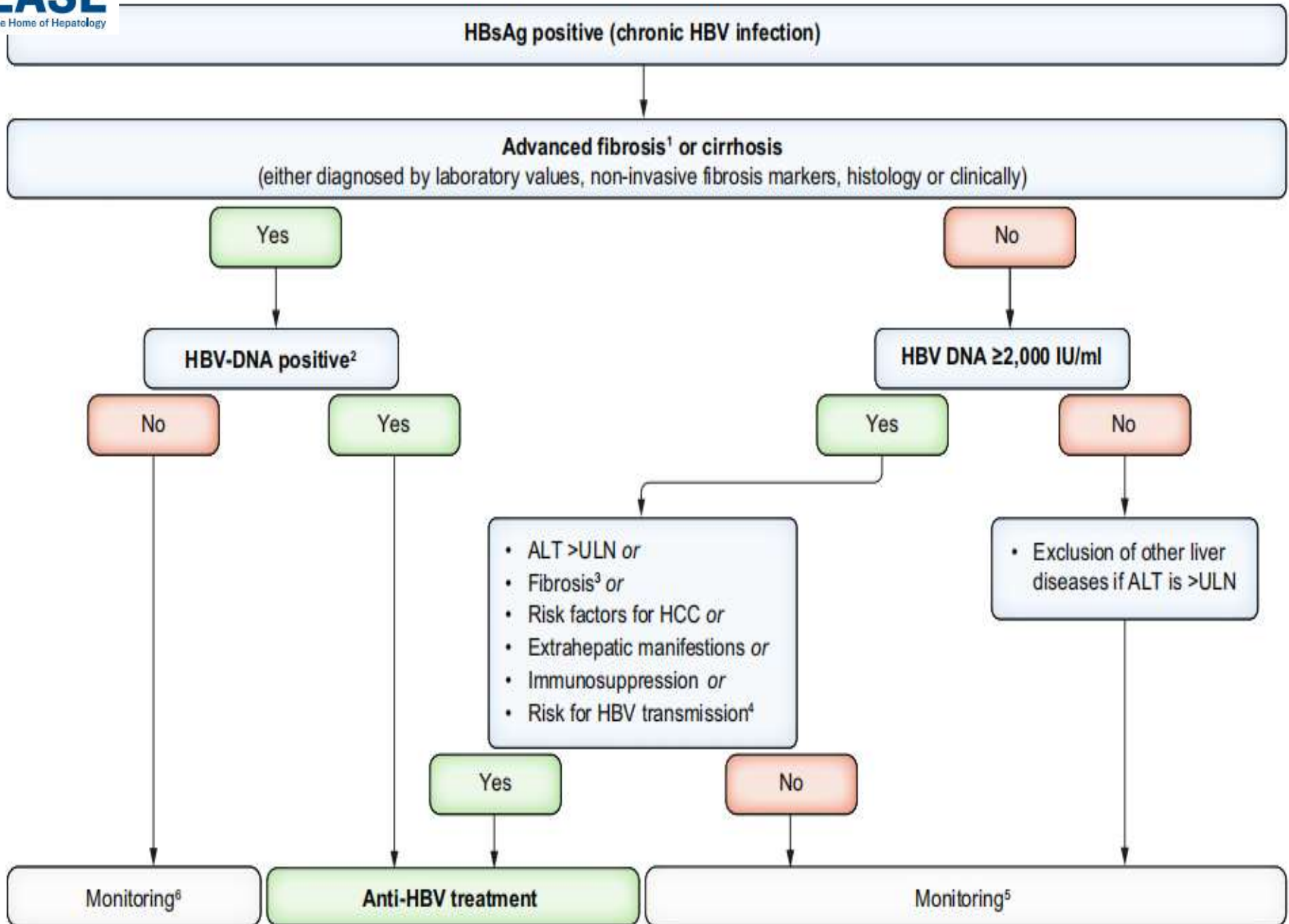
fonksiyon bozukluğu, DM,

yağlı karaciğer, metabolik

sendrom, kardiyak riskler

değerlendirilmeli

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Recommendations

- ETV, TDF, or TAF should be used as first-line NA therapy. When selecting between ETV, TDF and TAF, comorbidities (especially renal insufficiency and reduction in bone density) and concomitant circumstances (e.g. women of child-bearing age, pregnancy, age) as well as previous therapies should be taken into account (**LoE 1, strong recommendation, strong consensus**).
- Kidney function should be assessed before treatment initiation and monitored regularly during treatment to adjust the NA dose (**LoE 1, strong recommendation, strong consensus**).
- Treatment with TDF should be switched to another NA (ETV or TAF) if the glomerular filtration rate decreases, if tubulopathy occurs, and in case of hypophosphatemia or osteoporosis. Previous therapies and resistance should be taken into account when choosing the NA (**LoE 1, strong recommendation, strong consensus**).

“Hepatit B tedavisinde hastalardaki riskler değerlendirilmelidir”

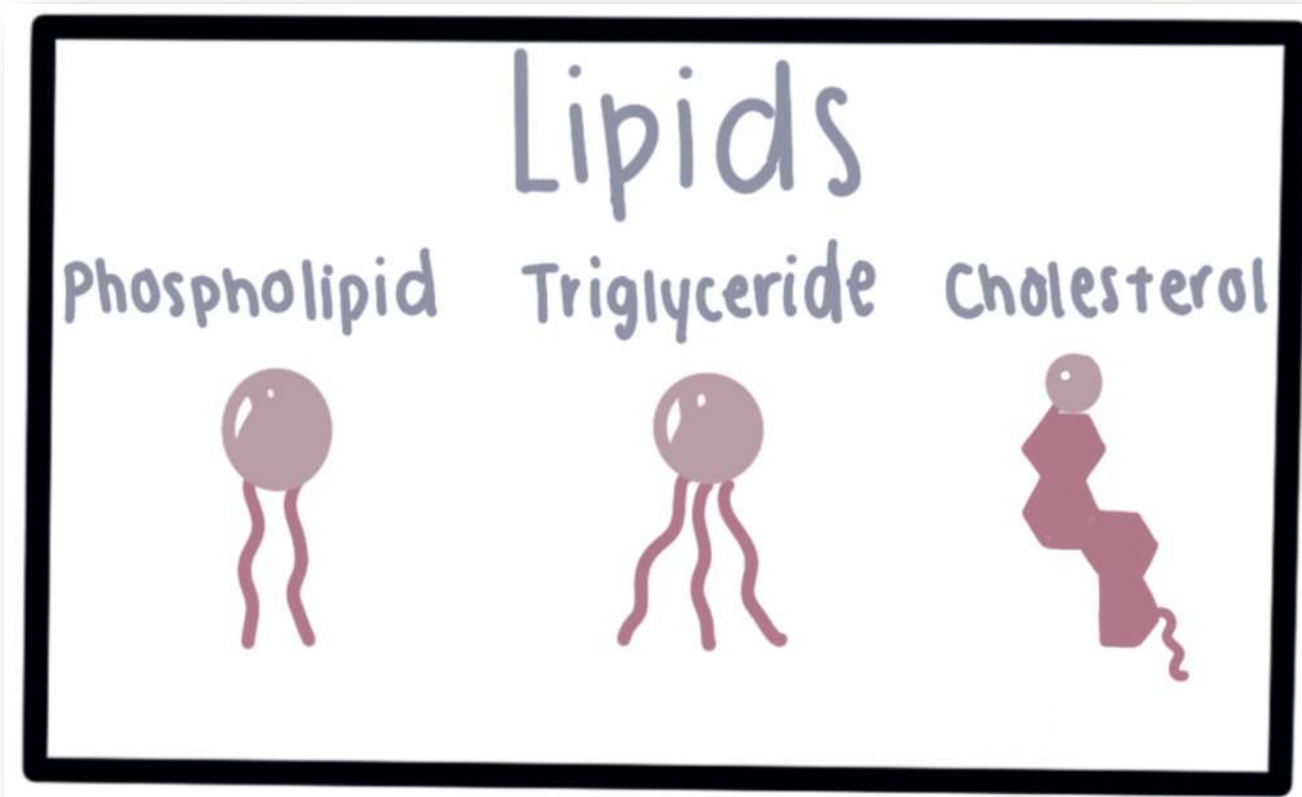
- ❖ Çalışmalar ve meta-analizler, **TAF içeren tedavi rejimlerinin, HIV ile yaşayan kişilerde ve hepatit B hastalarında TDF içeren rejimlere kıyasla daha yüksek lipid düzeyleriyle ilişkili olduğunu** ortaya koymuştur
- ❖ **TDF’den TAF’ye geçiş yapılan hastalarda, özellikle önceden mevcut metabolik riskleri olanlarda, lipid profillerinin yakından izlenmesi ve kardiyovasküler riskin dikkate alınması gereklidir.**





Table 8. Risk factors that have been associated with HCC risk in individuals with chronic HBV infection.

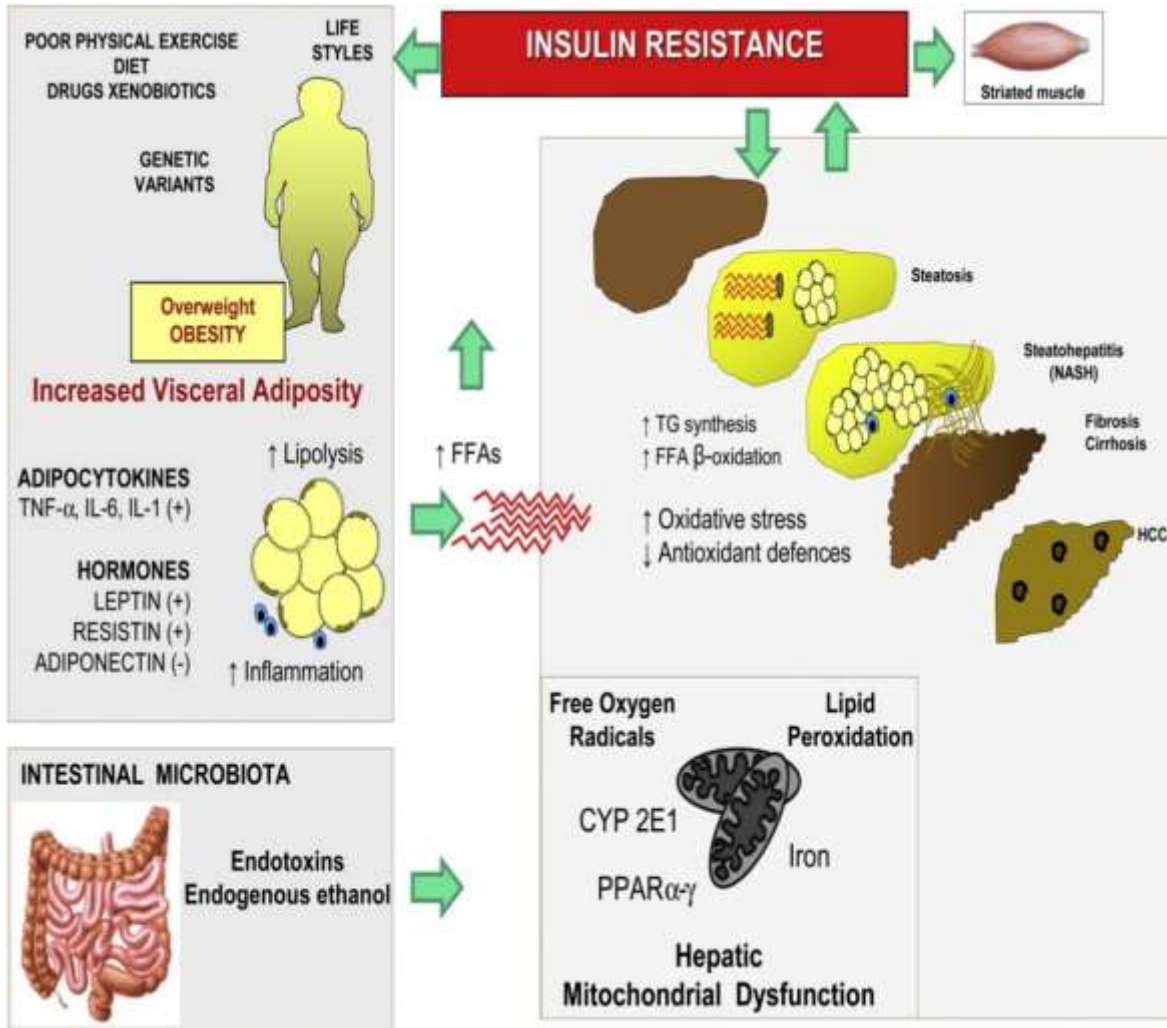
Risk factor	Comments and references
Viral factors	
HBV DNA*	Non-linear risk starting with >2,000 IU/ml ^{111,161,163} Highest risk in HBeAg-positive individuals with 6-7 log ₁₀ IU/ml (lower risk if HBV DNA is ≥8 log ₁₀) ²⁰⁷
HBSAg	High HBSAg (≥1,000 IU/ml) in HBeAg-negative individuals ¹⁶³
HBeAg*	In overall analyses, positive HBeAg (in individuals older than 30 years) is associated with HCC ^{163,166,167}
HBcrAg	Prognostic marker for occurrence and recurrence; ^{164,165,208} importance of HBcrAg in HBeAg-negative infection ²⁰⁹
HBV genotype	Genotype C, ¹⁹⁷ genotype A (e.g. in The Gambia ¹⁹⁹), genotype F in Alaska native persons, ¹⁹⁷ genotype D in India ²¹⁰
Core promoter mutations	Present ^{169,211}
Viral coinfections	HDV, ^{212,213} HCV, ^{167,184,214} HIV ²¹⁵
Host factors	
Cirrhosis	Strongest risk factor for HCC ^{161,167,169} HCC risk remains after viral suppression ^{170,171}
Low platelets*	Indicator for cirrhosis ¹⁹⁵
Family history of HCC	Independent risk factor in all phases of chronic HBV infection ^{192,193,216}
Age*	HCC risk increases with age, with most studies focusing on individuals older than 30 years. ^{161,185} Evidence increases with age ≥35, ¹⁶⁶ ≥40, ¹⁶³ ≥50. ¹⁶⁷ HCC risk varies in different age groups for men and women and for different ethnic groups ¹⁹¹
Sex*	Higher risk among males ^{161,166,169,185}
ALT	Elevated (or in the upper normal range) ^{162,166,167,185}
Type 2 diabetes mellitus (T2D)	T2D is independently associated with HCC. ^{183,217,218} Glycaemic burden is associated with HCC. ²¹⁹ T2D is included in HCC risk scores ^{168,218} However, one analysis showed that T2D was not independently associated with HCC in chronic HBV infection ¹⁷⁹
Steatotic liver disease (SLD)	Conflicting data: - Increased risk of HCC and cirrhosis ¹⁸⁸ - Lower risk of HCC, cirrhosis, and mortality ^{180,182,189}
Body mass index (BMI)	High BMI ≥30, ¹⁷⁹ HR stronger in females ¹⁷⁸
Metabolic syndrome	Multiple (≥3) metabolic risk factors or increasing burden of metabolic dysfunction are associated with HCC ^{180-182,190}
Cigarette smoking	Present ^{181,184-187}
Alcohol consumption	Heavy alcohol intake ≥60 g/d ¹⁶¹
Ethnicity	Evidence low or absent: - Birth in Africa/Oceania: linked to very early-onset HCC ¹⁹⁴ - Sub-Saharan Africans with HBV in Europe: lower HCC incidence, similar risk factors to general population ¹⁹⁵ - Western vs. Eastern studies: no significant age-adjusted differences in HCC incidence ¹⁶¹
Environmental factors	
Aflatoxin B1 (AFB1)	In high-exposure areas, AFB1 and HBV synergistically increase HCC risk; reducing aflatoxin exposure could lower HCC cases by 23% ¹⁹⁸
Air pollution	Association between fine particulate matter and HCC ^{200,201}

Hepatit B - Lipid metabolizması arasında bir ilişki var mı?



Nonalcoholic fatty liver disease

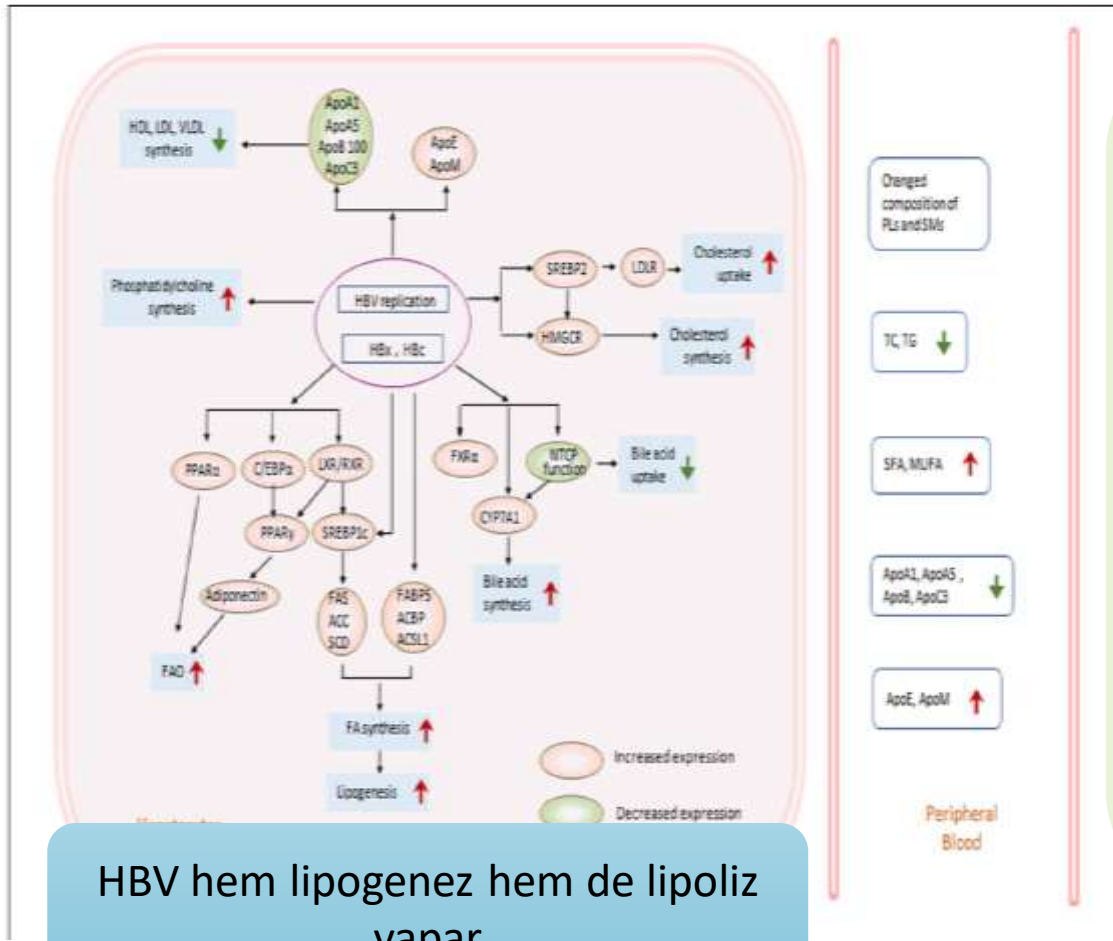
Marcin Krawczyk MD (Research Fellow)^a, Leonilde Bonfrate MD (Research Fellow)^b, Piero Portincasa MD, PhD (Associate Professor of Internal Medicine)^b  



*KC lipid üretilen organ
*Adipositokinlerden
Visfatin bakılmış HCV ile
anlamalı ilişki varken HBV
ile ilişki gösterilmemiş

Multifaceted Interaction Between Hepatitis B Virus Infection and Lipid Metabolism in Hepatocytes: A Potential Target of Antiviral Therapy for Chronic Hepatitis B

Jiaxuan Zhang, Ning Ling, Yu Lei, Mingli Peng, Peng Hu and Min Chen*



HBV hem lipogenez hem de lipoliz yapar

HBV "METABOLİK VİRUS"
METABOLOVIRUS

1. LDL R'ünü ve kolesterol alımı ve sentezi artar
2. Lipid emilimi için gerekli olan safra asid sentezini arttırır
3. Yağ asit sentezi artar
4. Bazı lipoproteinleri düşürürken bazılarının sentezini stimüle eder

Front Microbiol. 2021; 12:636897

Impact of hepatitis B virus infection on hepatic metabolic signaling pathway

Yi-Xian Shi, Chen-Jie Huang, Zheng-Gang Yang

Shi YX et al. Metabolic response to HBV infection

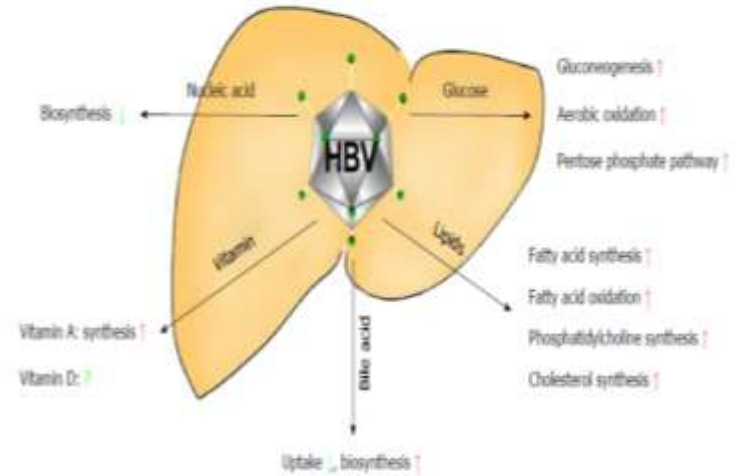


Figure 2 Changes in the hepatic metabolic signaling pathway induced by hepatitis B virus infection. Alterations in related signaling pathways (including glucose, lipids, nucleic acids, bile acids and vitamins) following hepatitis B virus (HBV) infection are marked and highlighted in this figure. The influence of HBV infection on vitamin D metabolism is unclear.

HBx proteini ile **lipid sentezi ve hepatosite yağ asit bağlanması** artarken

apolipoprotein salgılanması engellenir

Böylece periferik kanda Kolesterol ve Triglicerid azalır, LDL, VLDL çok düşer.

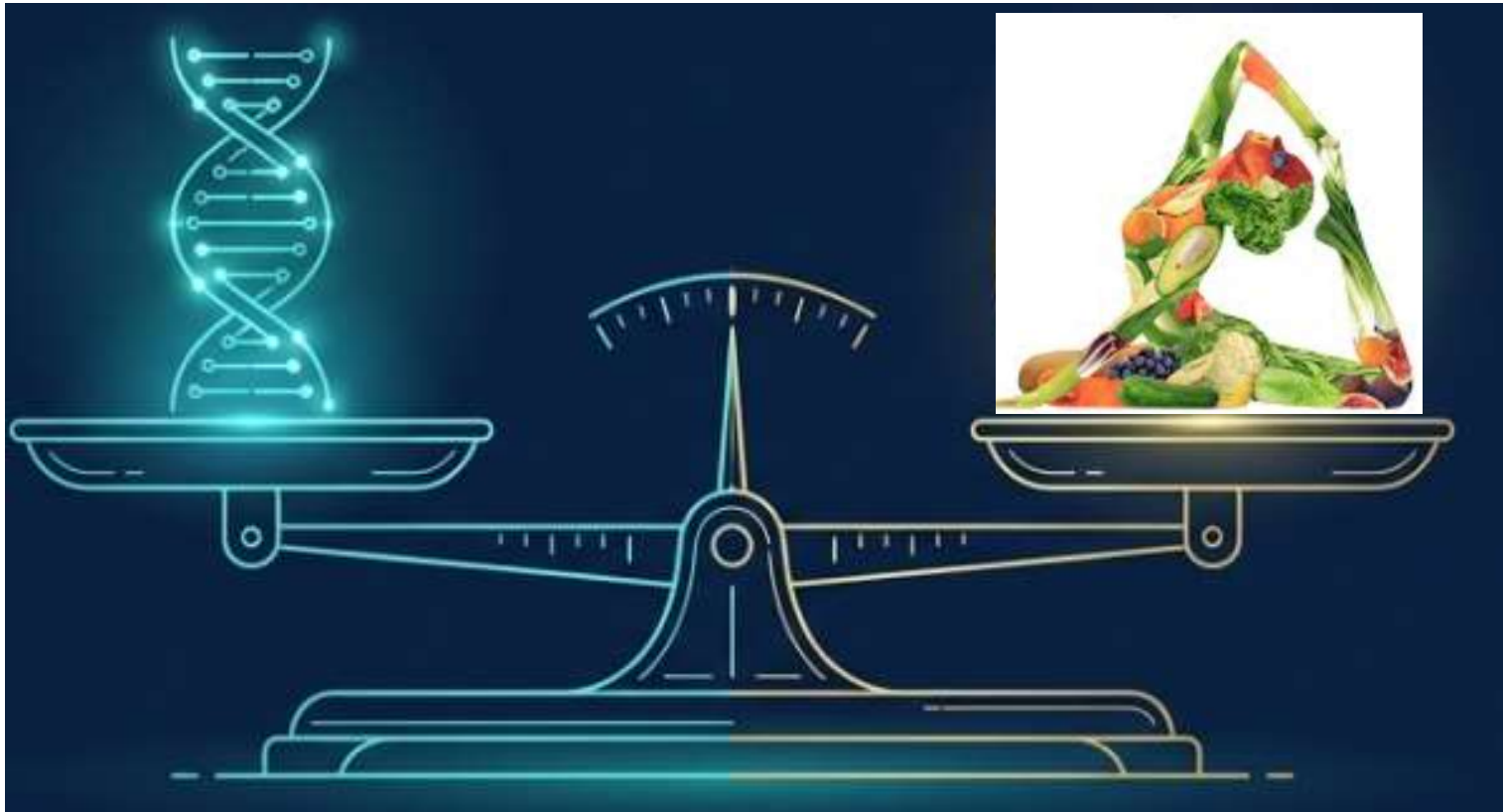
Ancak **karaciğer yağlanması** artar

Metabolik sađlık

Bel evresi, alık kan Őekeri, sistolik kan basıncı, **trigliseridler** ve **HDL kolesterol** dahil olmak üzere beŐ temel lmün sađlıklı aralıkta olması olarak tanımlanır.

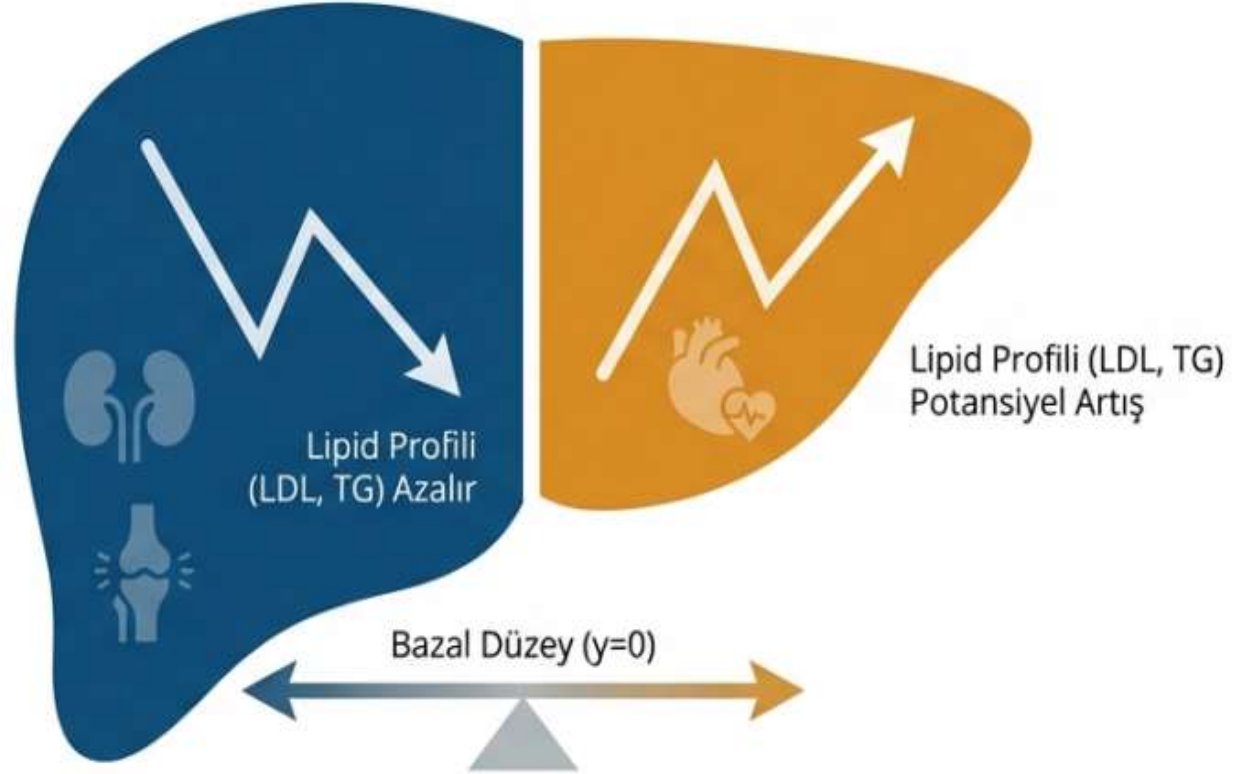


Hepatit B Tedavisi ve Antiviral Dengesi



Kronik Hepatit B Yönetiminde Klinik Denge

Birinci Basamak Antivirallerde (TDF vs. TAF) Metabolik ve Kardiyovasküler Ayrım



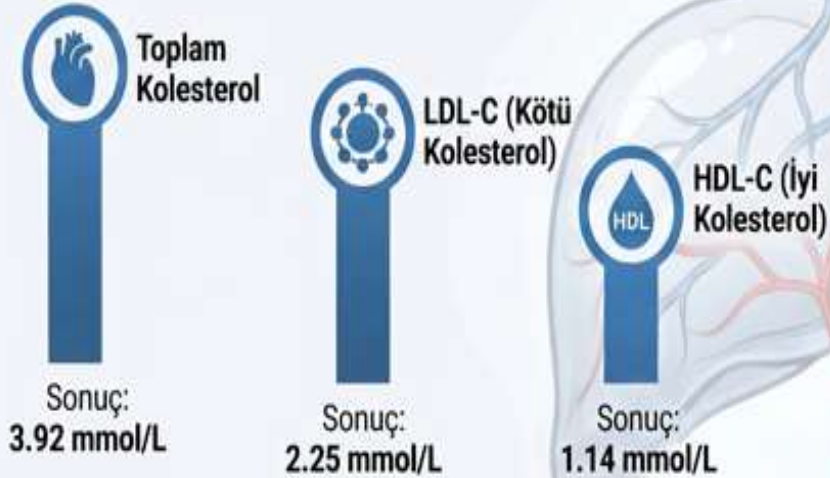
Yüksek genetik bariyerli NUC'lar ile viral süpresyon çözülmüş bir problemdir. Yeni klinik odak noktası, ömür boyu sürecek tedavilerde **kişiselleştirilmiş metabolik ve sistemik risk yönetimidir.**

Kronik Hepatit B Tedavisinde TDF ve Entekavir: Lipid Profili Karşılaştırması

Bu çalışma, HIV negatif 348 Kronik Hepatit B taşıyıcısında TDF ve Entekavir'in (ETV) lipid seviyeleri üzerindeki etkilerini karşılaştırmaktadır. TDF'nin kalp damar hastalığı riskini etkileyebilecek şekilde lipid seviyelerini önemli ölçüde düşürdüğü bulunmuştur.

TDF ile Kolesterolde Belirgin Düşüş

TDF kullanan hastalarda toplam kolesterol, LDL ve HDL seviyelerinde anlamlı azalma görülmüştür.



Entekavir Grubunda Değişim Gözlenmedi

ETV ile tedavi edilen hastalarda lipid profillerinde tedavi öncesine göre anlamlı fark yoktur.



%20 Daha Fazla Azalma Olasılığı:
TDF hastalarının lipid seviyelerinde %20 düşüş yaşama olasılığı ETV'ye göre belirgin düzeyde yüksektir.

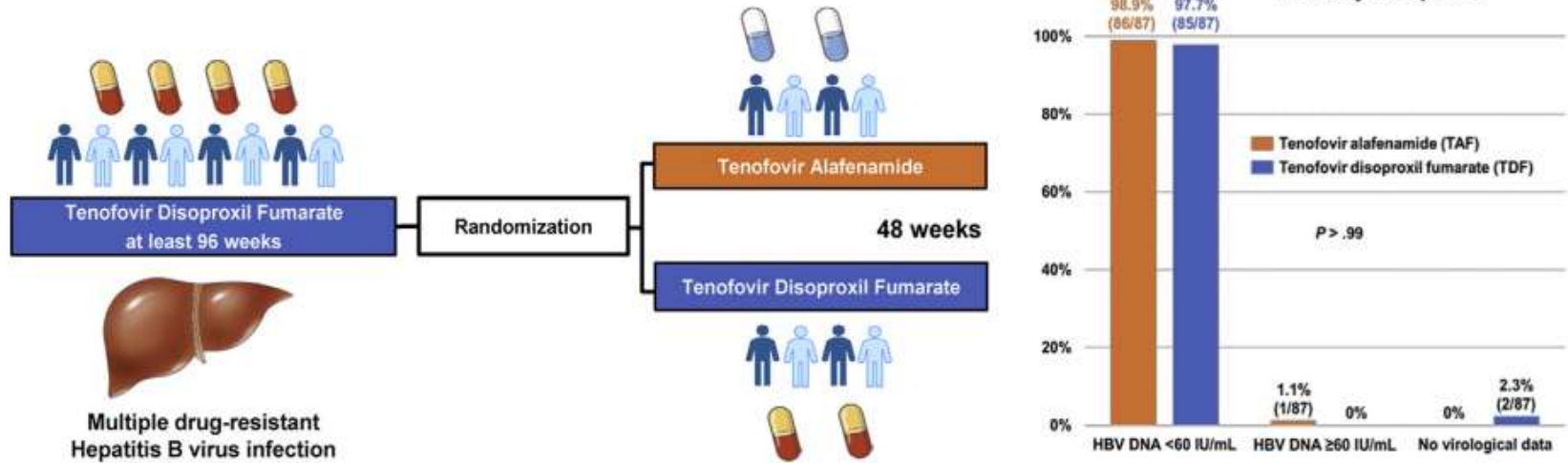
Klinik Çıkarımlar ve Sonuç

- Kardiyovasküler Risk Yönetimi:** TDF'nin lipid düşürücü etkisi, kalp damar hastalığı riski olan CHB hastalarında ilaç seçimini etkileyebilir.
- Trigliserid Seviyeleri Sabit Kaldı:** Her iki tedavi grubunda da trigliserid seviyelerinde anlamlı bir değişiklik gözlemlenmemiştir.

Shaheen AA, et al. *Aliment Pharmacol Ther.* 2017;1–6.

Tenofovir Alafenamide for Drug-Resistant Hepatitis B: A Randomized Trial for Switching From Tenofovir Disoproxil Fumarate

Kwan Soo Byun,^{*,a} Jonggi Choi,^{‡,a} Ji-Hoon Kim,^{*} Yung Sang Lee,[‡] Han Chu Lee,[‡] Yoon Jun Kim,[§] Byung Chul Yoo,^{||} So Young Kwon,^{||} Geum-Youn Gwak,^{||} and Young-Suk Lim[‡]



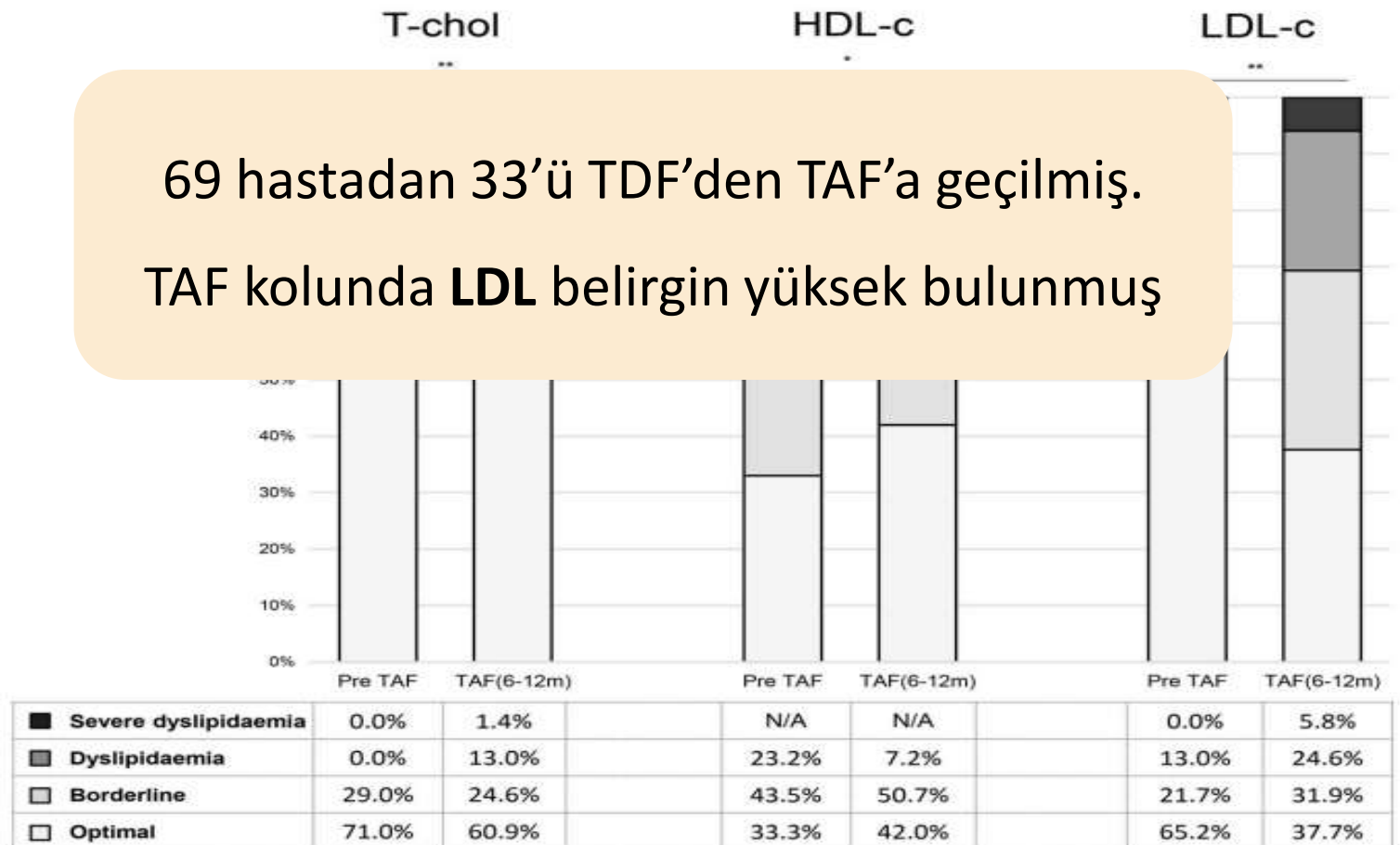
- 84 TAF/80 TDF 48 hafta takip **etkililik** açısından **fark yok**
- TAF alanlarda **LDL** belirgin yüksek/
- TAF kanadında **kilo alımı daha fazla**

Effect of switching from tenofovir disoproxil fumarate to tenofovir alafenamide on lipid profiles in patients with hepatitis B

PLoS ONE. 2022;17(1): e0261760.

Kazuharu Suzuki^{1,2}, Goki Suda^{1*}, Yoshiya Yamamoto², Satoshi Abiko²,

69 hastadan 33'ü TDF'den TAF'a geçilmiş.
TAF kolunda **LDL** belirgin yüksek bulunmuş

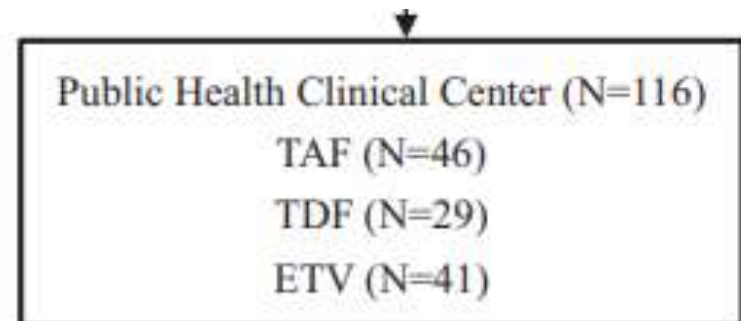
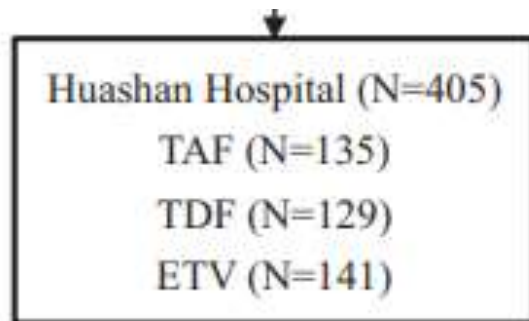


Effect of nucleos(t)ide analogues on blood lipid profiles in patients with chronic hepatitis B

A cross-sectional survey

Jing Wen Chen, BS^a, Xiong Yue Cao, BS^b, Xun Qi, PhD^b, Ji Ming Zhang, MD, PhD^{a,*}

Chen et al. *Medicine*. 2022; 101:50



Effect of nucleos(t)ide analogues on blood lipid profiles in patients with chronic hepatitis B

A cross-sectional survey

Chen et al. *Medicine*. 2022; 101:50

Table 5

Comparison in lipid profiles between patients treated with TAF, TDF and ETV before and after NAs treatment.

						TDF vs. ETV
<p>❖ The blood lipid levels in the TDF group were lower than those in the TAF group and ETV group, and the occurrence of hyperlipidemia was associated with age, sex, BMI, and different treatment.</p>						
Total number, n (%)						
Baseline lipid profile						
TC (mmol/L)						.044
TG (mmol/L)						.351
HDL-C (mmol/L)						.983
LDL-C (mmol/L)						.298
VLDL-C (mmol/L)						.019
APoA1 (mmol/L)						.129
APoB (mmol/L)						.209
Lipid profile after NA treatment						
TC (mmol/L)	4.60 ± 1.01	3.84 ± 0.69	4.49 ± 0.90	.003	.374	.065
TG (mmol/L)	0.99 ± 0.46	0.76 ± 0.44	1.09 ± 0.72	.771	1.000	.934
HDL-C (mmol/L)	1.43 ± 0.39	1.23 ± 0.21	1.34 ± 0.29	.451	1.000	.885
LDL-C (mmol/L)	2.68 ± 0.73	2.23 ± 0.54	2.60 ± 0.89	.017	.294	.234
VLDL-C (mmol/L)	0.49 ± 0.20	0.38 ± 0.16	0.54 ± 0.24	.128	.752	.107
APoA1 (mmol/L)	1.00 ± 0.16	0.91 ± 0.10	0.98 ± 0.10	.047	.712	.137
APoB (mmol/L)	0.53 ± 0.13	0.45 ± 0.12	0.53 ± 0.16	.068	.978	.101

APoA1 = apolipoprotein A1, APoB = apolipoprotein B, ETV = entecavir, HDL-C = high-density lipoprotein cholesterol, LDL-C = low-density lipoprotein cholesterol, NAs = nucleos(t)ide analogues, TAF = tenofovir alafenamide fumarate, TC = total cholesterol, TDF = tenofovir disoproxil fumarate, TG = triglyceride, VLDL-C = very low-density lipoprotein cholesterol.

Lipid safety of tenofovir alafenamide during 96-week treatment in treatment-naive chronic hepatitis B patients

Wenjuan Zhao¹, Yi Liu¹, Mengdi Zhang², Zixin Cui¹, Zhan Qu¹, Yiyang Li¹, Meijuan Wan¹, Wen Wang¹, Yunru Chen¹, Lei Shi¹, Jianzhou Li¹ and Feng Ye^{1*}

Table 3 Impact of tenofovir alafenamide on achieving a higher level of total cholesterol in chronic hepatitis B patients

Characteristic	TCHO, increased change (%) by using TAF compared with ETV ^{1,2} , OR (95%CI)	P value
5% high		
10% high		
15% high		

TAF ve ETV alan hastalar

1 yıl sonunda TAF kolunda belirgin T kolesterol artışı olmuş

Effect of switching from prior Nucleos(t)ide Analogue(s) to Tenofovir alafenamide on lipid profile and cardiovascular risk in patients with Chronic Hepatitis B

Witchayaporn Praguylertluck^{1†}, Apichat Kaewdech^{2†}, Naichaya Chamroonkul², Teerha Piratvisuth^{2,3}, Pimsiri Sripongpun^{1c}^{2*}

Table 1. Baseline characteristics at the time of switch to TAF (N=110).

	ETV mono (N=22)	LAM mono (N=47)	TDF-based (N=41)	P value
Age, year, mean (SD)	53.05 (11.22)	55.11 (11.92)	59.46 (11.77)	0.080
Male sex, n (%)	15 (68.18)	26 (55.32)	24 (58.54)	0.596
Weight, kg, mean (SD)	65.35 (12.13)	63.14 (10.61)	64.23 (13.46)	0.766
BMI, kg/m ² , mean (SD)	24.02 (3.62)	24.26 (3.79)	24.56 (4.37)	0.867
<i>Thai CV risk</i>				
Risk score, median (IQR)	6.82 (2.43,17.24)	8.06 (3.98,13.75)	15.81(5.19,24.19)	0.029
CV Risk group				0.021
<10%	14 (63.64)	30 (63.83)	15 (37.5)	
10-20%	5 (22.73)	9 (19.15)	7 (17.5)	
>20%	3 (13.67)	8 (17.02)	18 (45)	

Table 1. Baseline characteristics at the time of switch to TAF (N=110).

	ETV mono (N=22)	LAM mono (N=47)	TDF-based (N=41)	P value
Changes of Laboratory data and characteristics				
Δ weight, kg, median(IQR)	0 (-1.3,0.85)	0 (-1,1.75)	1 (0,3)	0.010
Δ BMI, kg/m ² , median(IQR)	0 (-0.53,0.33)	0 (-0.45,0.64)	0.42 (0,1.28)	0.009
Δ LDL, mg%, median(IQR)	-2.45 (-13.15,10.93)	-5.9 (-16.55,9.9)	8.8 (2,19.8)	< 0.001
Treatment of dyslipidemia				
increase dose or potency of statin,n (%)	0 (0)	5 (10.64)	6 (14.63)	0.185
Thai CV risk				
Thai CV Risk score, median (IQR)	8.79 (2.4,18.00)	9.41 (3.74,18.05)	13.87 (7.31,24.37)	0.044
Thai CV Risk score change, median (IQR)	0.10 (-0.66,1.52)	0.51 (-0.38,2.70)	0.61 (-0.99,3.76)	0.544

Conclusion

Significant increase in LDL-c and total cholesterol after switching to TAF were observed only in patients with prior TDF, but not in those with prior ETV or LAM. Careful monitoring of lipids after the switch may not be universally needed. Data regarding long-term cardiovascular outcomes are warrant.

Kronik Hepatit B Tedavisinde TAF ve ETV: Serum Lipid Seviyeleri Üzerindeki Etkiler

336 KHB hastasının katıldığı retrospektif bir kohort çalışmasına dayanan analiz, birinci basamak anti-viral tedaviler olan TAF ve ETV'nin bir yıllık kullanım sonucunda lipid profilleri ve nonalkolik yağlı karaciğer hastalığı (NAFLD) riski üzerindeki etkilerini incelemektedir.

TAF (Tenofovir Alafenamid)

TAF Toplam Kolesterol (TCHO) Seviyelerini Artırıyor

TAF tedavisi alan hastalarda TCHO seviyeleri, bazal değerlere göre %5 oranında anlamlı artış göstermiştir.



+5%
TCHO Artışı

ETV Lipid Profili Üzerinde Nötr Etki Gösteriyor

Entekavir (ETV) grubu hastalarında bir yıllık tedavi sonrası lipid seviyelerinde anlamlı bir değişim gözlenmemiştir.



Nötr Etki
(Anlamlı Değişim Yok)

ETV (Entekavir)

Tedavi Sonrası TAF ve ETV Grupları Arasındaki Lipid Farkları (mmol/L)

Parametre	TAF Grubu (Post-Tx)	ETV Grubu (Post-Tx)	P Değeri
Toplam Kolesterol (TCHO)	4.67 ± 0.90	4.36 ± 1.05	0.006
Trigliserid (TG)	1.37 ± 0.81	1.24 ± 0.61	0.126
LDL Kolesterol	3.14 ± 0.92	3.15 ± 1.00	0.906

TAF Tedavisinde Kolesterol Artışı İçin Risk Faktörleri



Metabolik Faktörlerin Belirleyici Rolü

Vücut Kitle İndeksi (VKI) ve hipertansiyon, TCHO artışı ile doğrudan ilişkili temel risk faktörleridir.



Demografik ve Klinik Göstergeler

Erkek cinsiyet ve yüksek bazal TCHO seviyeleri, tedavi sırasında kolesterol yükselme olasılığını artırır.



CK-MB Enzimi ve Lipid İlişkisi

Kreatin kinaz-MB (CK-MB) izoenzim seviyeleri, lojistik regresyon analizinde yükselmiş TCHO ile ilişkili bulunmuştur.

NAFLD İnsidansında Kısa Vadeli Artış Saptanmadı:

TAF kaynaklı lipid artışına rağmen, bir yıllık süreçte yağlı karaciğer hastalığı riskinde artış görülmemiştir.

Metabolic effects and cardiovascular disease risks of TDF or TAF in patients with chronic hepatitis B: a systematic review and meta-analysis

Yuan-Hai Zhou^{1,2}, Nan Cai^{2,3}, Yu-Xin Chen^{2,3}, Yong-Lu Su^{2,3} and Peng Hu^{1,2,4*}

Front Pharmacol. **2025**; 16:1604972.

Meta-analizde toplam **19** çalışmadan **19.396** kronik **hepatit B**

- **12.067'si** sadece **TDF**, **5.423'** ü sadece **TAF** grubunda
- **1906'sı** **TDF'den TAF'ye geçiş** grubunda

in this meta-analysis. We found that both TAF and TDF treatment mildly increase the 10-year ASCVD risk. The TAF treatment showed significant increases in body weight, with no significant effects were observed on lipid levels or blood glucose.

While TDF treatment has a lipid-lowering effect and caused weight loss.

Subanalyses emphasized the impact of changing antiviral treatment strategies

on metabolism. We found an increased risk of dyslipidemia and body weight gain after switching from TDF to TAF treatment.

Metabolic effects and cardiovascular disease risks of TDF or TAF in patients with

Author, year	Study design	Number of patients	Treatment strategy	Main conclusion
Cheng et al. (2024)	[PC]	TDF: 99 TAF: 99	Switch (TDF→TAF)	the ASCVD risk did not enhance in TDF-switched patients. The switch from TDF to TAF significantly increased body weight, triglyceride, total cholesterol, HDL, LDL, fasting glucose, insulin and insulin Resistance
Ogawa et al. (2022)	[RC]	TDF: 116 TAF: 116	Switch (TDF→TAF)	total, low-density lipoprotein high-density lipoprotein cholesterol and triglycerides were significantly increased after the switch to TAF. But the total to HDL cholesterol ratio remained unchanged
Yeh et al. (2022)	[RC-PSM]	TDF: 91 TAF: 91	Switch (TDF→TAF)	In real-world NUC-experienced CHB patients, unexpected body weight gain was observed after TAF switching
Akdemir Kalkan et al. (2022)	[RC]	TDF: 237 TAF: 237	Switch (TDF→TAF)	low-density lipoproteins cholesterol was observed to be significantly higher after 6 months compared to baseline values And the TC increased significantly in the TAF group

Front Pharmacol. **2025**; 16:1604972.

Metabolic effects and cardiovascular disease risks of TDF or TAF in patients with chronic hepatitis B: a systematic review and meta-analysis

Yuan-Hai Zhou^{1,2}, Nan Cai^{2,3}, Yu-Xin Chen^{2,3}, Yong-Lu Su^{2,3} and Peng Hu^{1,2,4*}

TABLE 3 The characteristics of patients

Treatment status					p-value
TDF→TAF	TDF (mg/dL)	0	4.17	2.17 to 6.17	00.770
	LDL (mg/dL)	6	11.13	7.81 to 14.45	59.1%
	TC/HDL (mg/dL)	4	0.04	-0.02 to 0.1	0.0%
	Body weight Change (kg)	3	0.73	0.23 to 1.2	0.0%

Sonuç: TAF ve TDF tedavileri farklı metabolik özellikler göstermektedir. Kardiyovasküler hastalık riskleri ve kilo alımı açısından hastalar yakın takip edilmelidir.

Long-term efficacy and safety of nucleos(t)ides analogues in patients with chronic hepatitis B

Ther Adv Infectious Dis. 2021; 8: 1–13

Luisa Roade , Mar Riveiro-Barciela, Rafael Esteban and Maria Buti

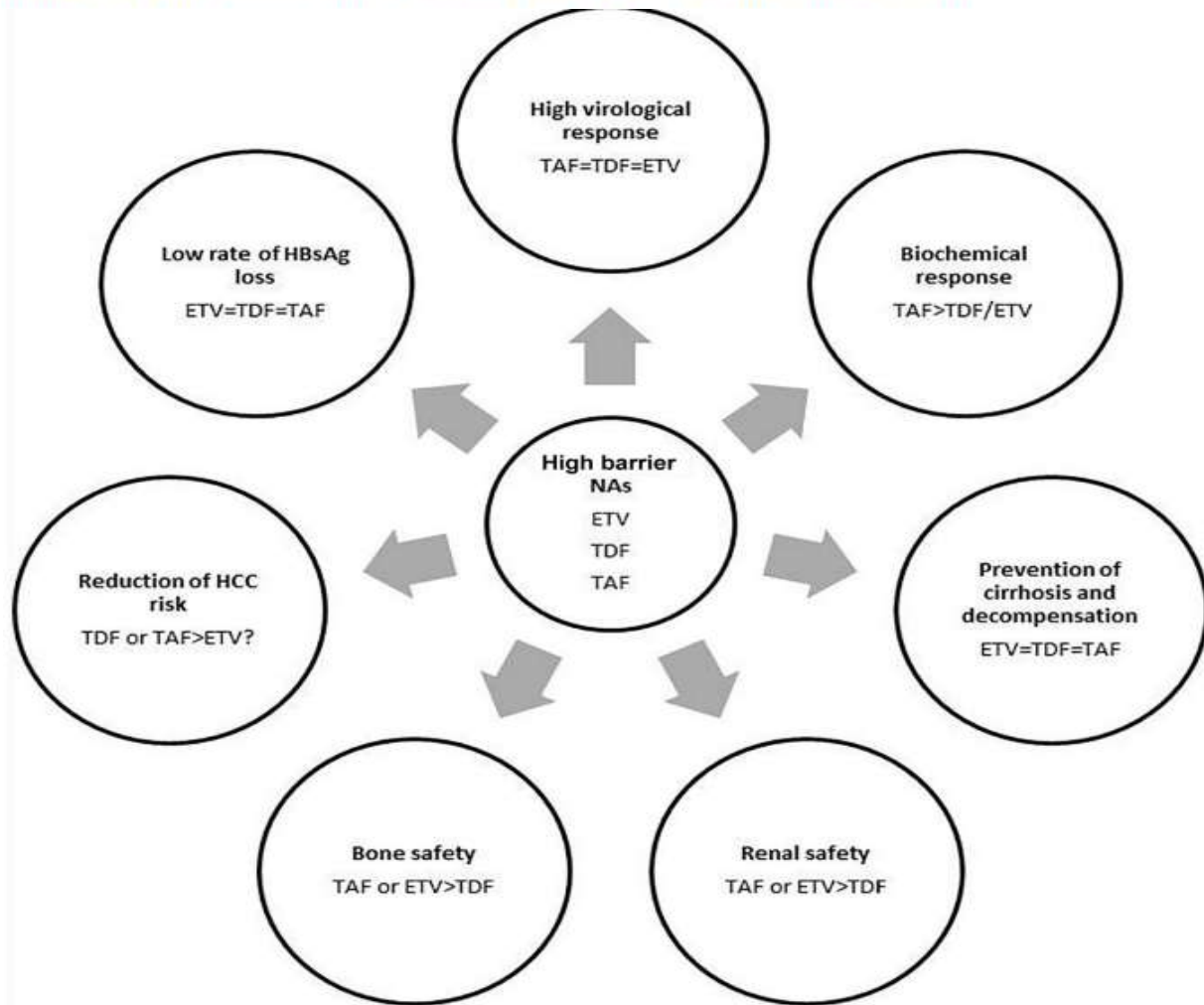






Table 1. Monitoring recommendations and considerations for first line NAs.

	TDF	TAF	ETV
 böbrek	<p>Monitor with serum creatinine, eGFR and serum P⁺ at start and then regularly.</p> <p>Dose adjustment if eGFR <50 ml/min per 1.73 m².</p> <p>Consider switch to TAF or ETV if P⁺ <2.5 mmol/dL, eGFR <60 ml/min per 1.73 m² or elderly.⁷³</p>	<p>No monitoring needed. No dose adjustment needed.</p> <p>Scarce evidence in hemodialysis and eGFR <15 ml/min per 1.73 m².</p> <p>Favorable preliminary data.^{71,72}</p>	<p>Monitor with serum creatinine and eGFR at start and then regularly.</p> <p>Dose adjustment if eGFR <50 ml/min per 1.73 m².</p>
Lipid			
 kemik	<p>Consider switch to TAF or ETV if P⁺ <2.5 mmol/dL, concomitant bone condition or elderly.</p>	<p>No monitoring needed.</p>	<p>No monitoring needed.</p>
 İleri evre kc	<p>Usual dose. Extreme kidney monitoring.</p>	<p>Scarce evidence. Favorable preliminary data.⁷³</p>	<p>Increase usual dose to 1 mg/day. Extreme kidney monitoring.</p>
 İlaç direnci	<p>Extremely infrequent. Ensure adherence. Switch to ETV or combination therapy.</p>	<p>Not reported.</p>	<p>Resistance in 1% in naïve, 50% LMV-experienced. Ensure adherence. Switch to TDF usual dose in monotherapy.</p>
 gebelik	<p>Recommended.</p>	<p>Scarce evidence. Limited favorable data in HIV mono-infected women.⁷⁴</p>	<p>Not recommended.</p>
 >2 yaş	<p>Above 2 years old.</p>	<p>Scarce experience, above 12 years old.</p>	<p>Above 2 years old.</p>
 HIV koenfeksiyon	<p>As part of HAART.</p>	<p>As part of HAART.</p>	<p>Not recommended in monotherapy.</p>
 HDV koenfeksiyonu	<p>No direct anti-HDV activity.⁷⁵ Might be added to peg-IFNα and/or new therapies according to HBV replication.⁷⁶</p>	<p>No direct anti-HDV activity. Data extrapolated from other NAs.⁷⁵ Might be added to peg-IFNα and/or new therapies according to HBV replication.⁷⁶</p>	<p>No direct anti-HDV activity.⁷⁵ Might be added to peg-IFNα and/or new therapies according to HBV replication.⁷⁶</p>

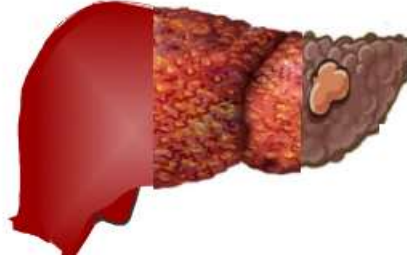
Sonuç olarak;

KHB hastalarının yönetimi ve tedavisi: **Kişiselleştirilmiş tıbbı doğru**

HBV cure vs
HBV infection control



Liver disease cure vs control



- ✓ Hepatit B tedavisinde direnç bariyeri **ETV, TDF ve TAF** kullanılmaktadır
- ✓ Antiviral seçiminde **kardiyak riskler, metabolik durum, kan lipid sevipleri** değerlendirilmelidir.
- ✓ Hepatit B, güvenli, mevcut ve etkili **aşılarla** önlenabilir.





Emek ve dayanışma günümüz kutlu olsun

