

# **Diabetik Hastalarda Non- diabetik Glomerüler Böbrek Hastalığı**

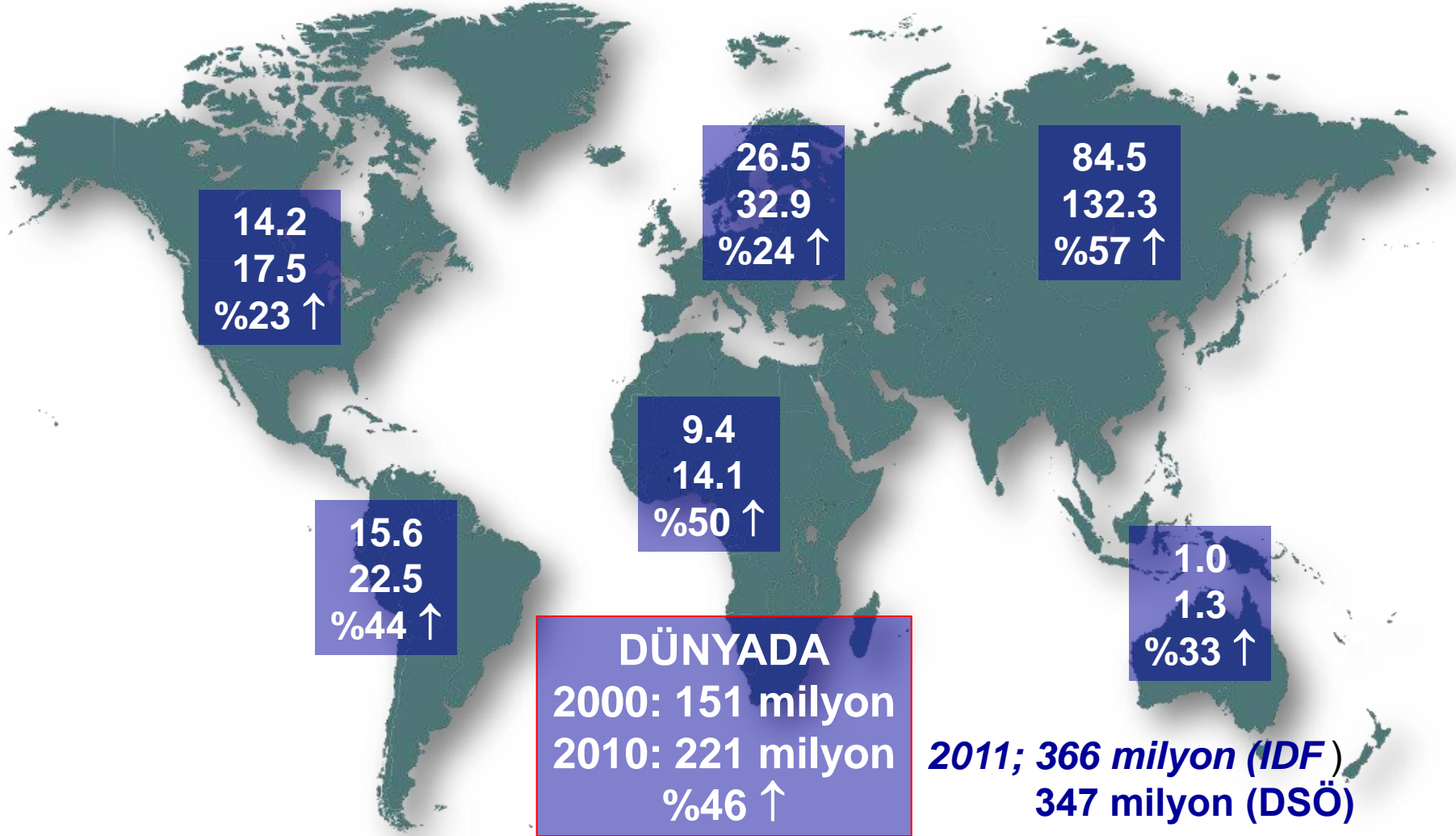
**Prof. Dr. Gültekin Süleymanlar  
Akdeniz Üniversitesi Tıp Fakültesi  
Nefroloji BD, Antalya**

**51. Ulusal Diabet Kongresi  
22-26 Nisan 2015  
Sungate Rixos Hotel, Beldibi Antalya**

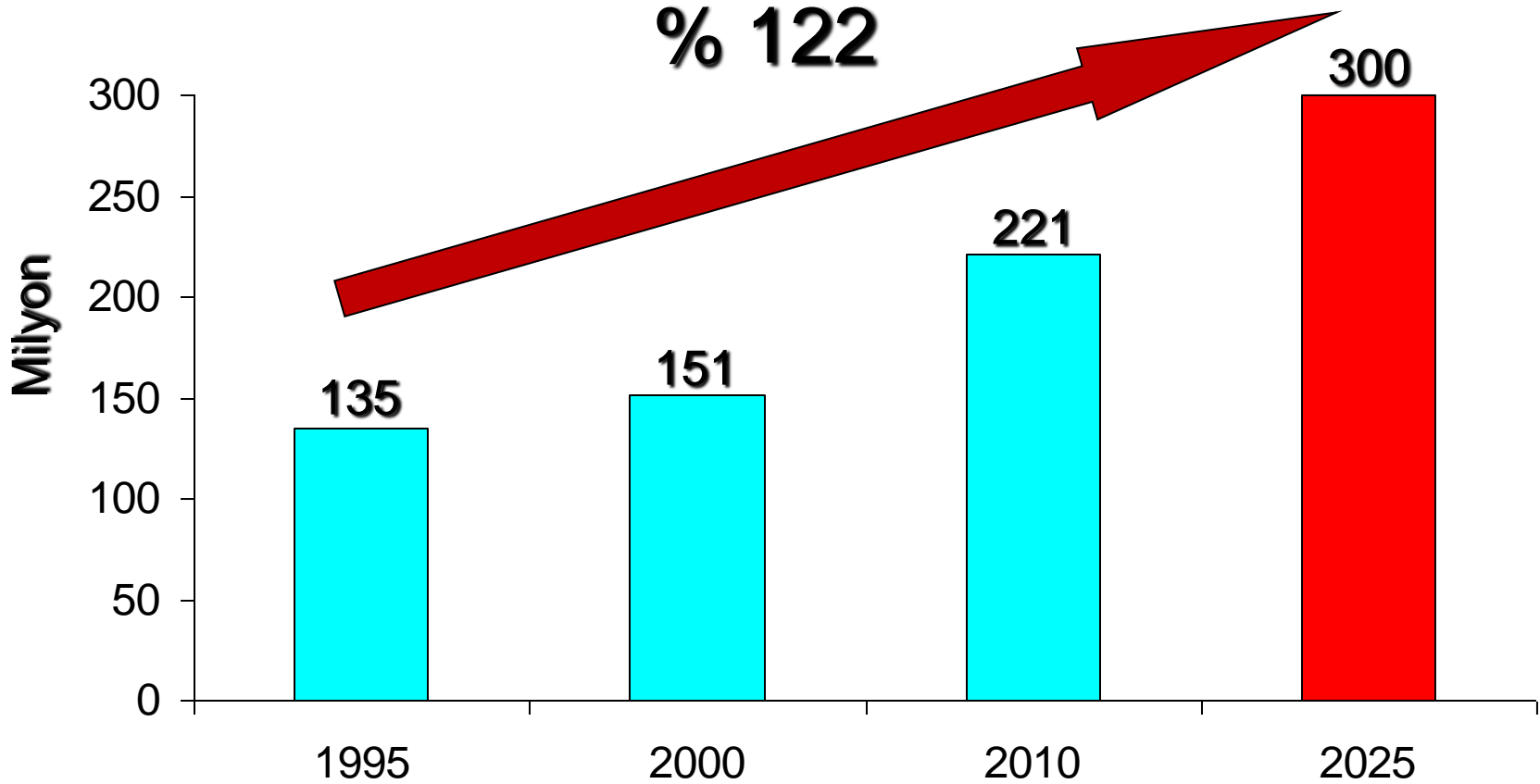
In order to be a competent nephrologist,  
one has to be a knowledgeable diabetologist

*Eli Friedman*

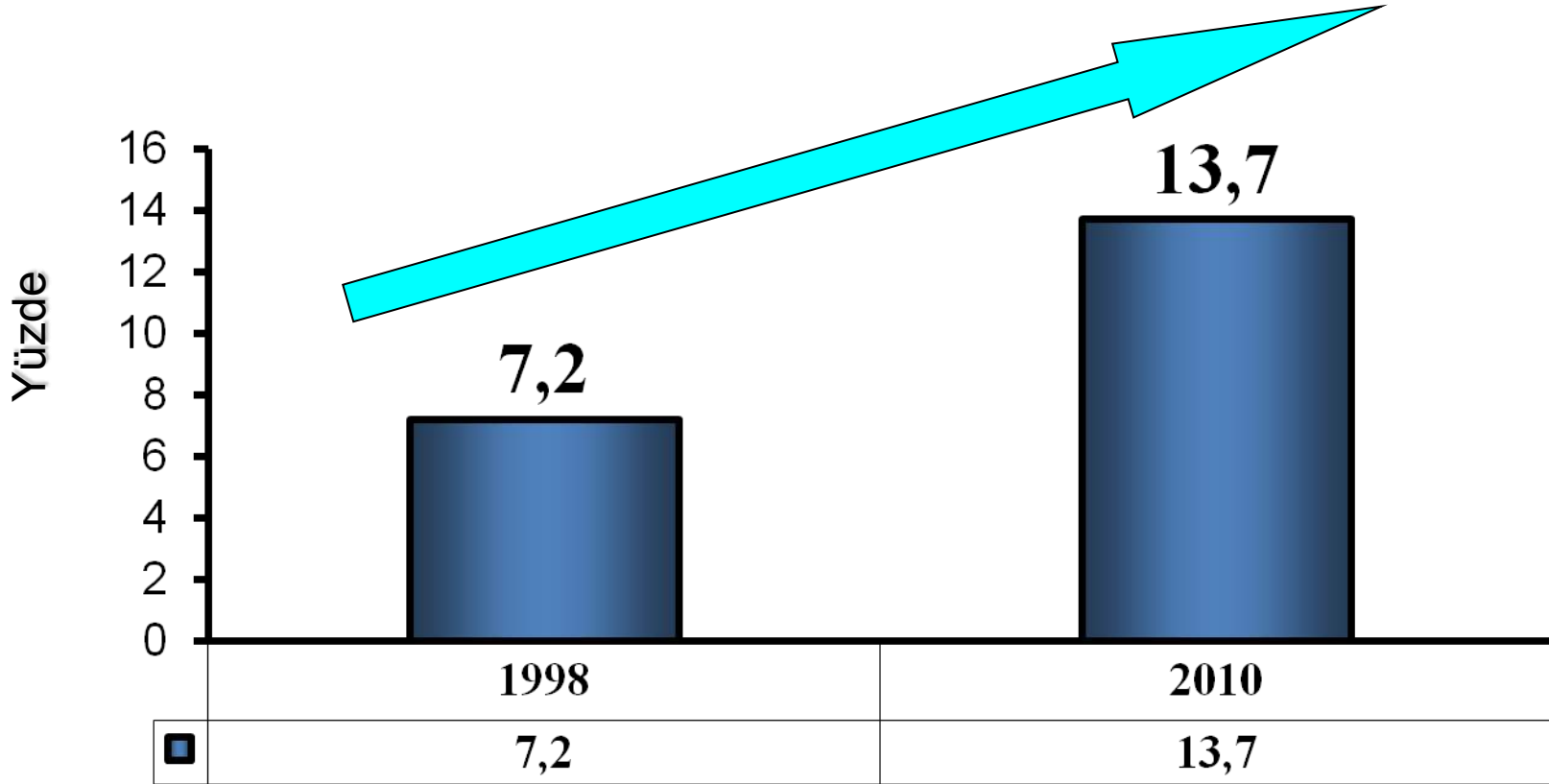
# Dünyada Diyabet Sıklığı



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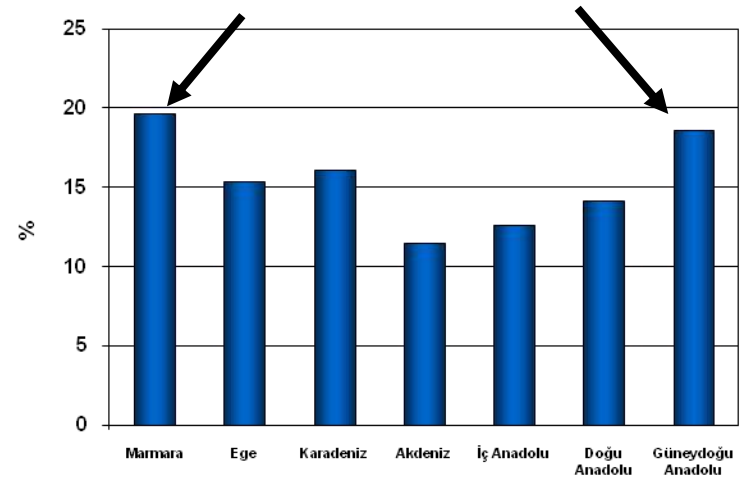
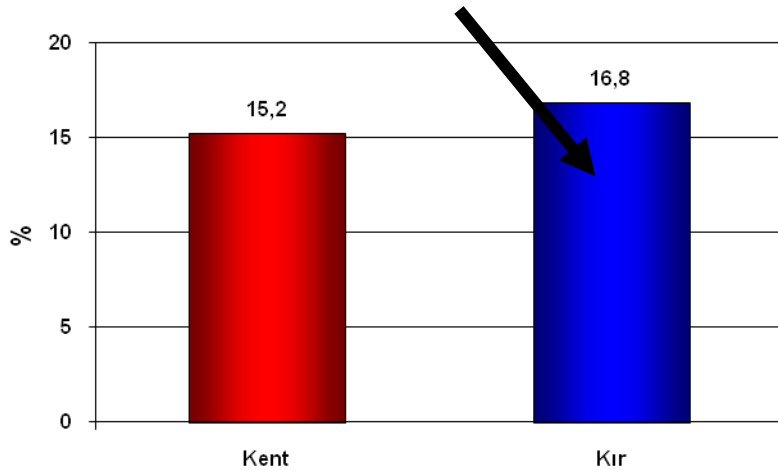
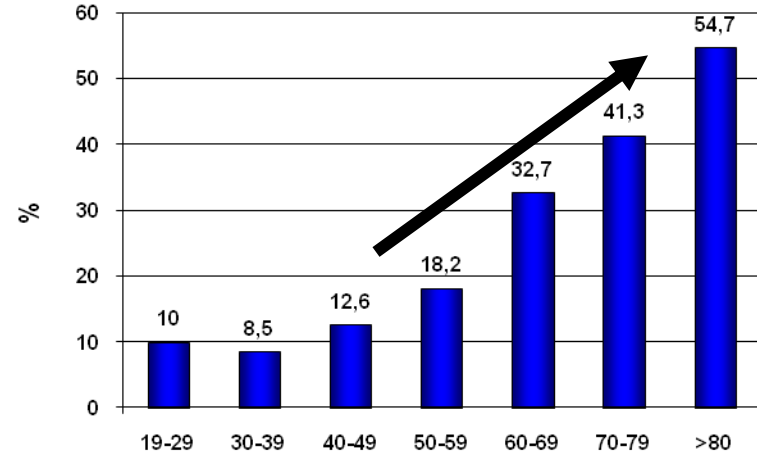
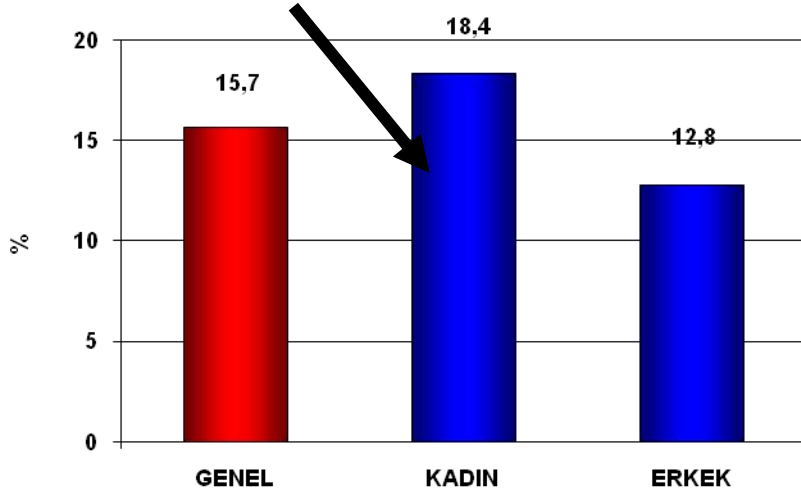
# Türkiye'de Diabet Sıklığı



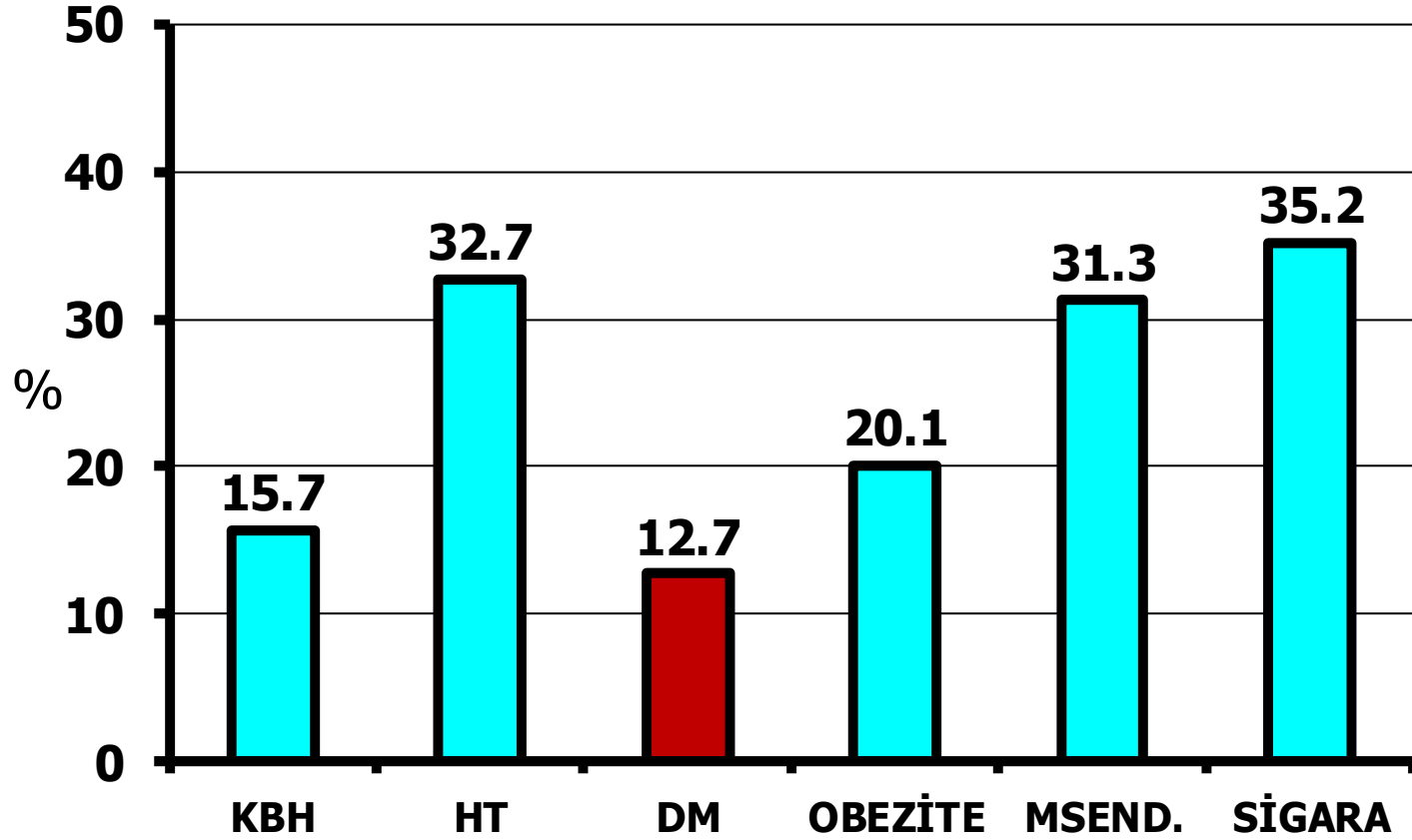
TURDEP-I: Satman I, et al. Diabetes Care 2002;25:1551-6  
TURDEP-II

# Kronik Böbrek Hastalığı-TR

(Cinse, yaşa, yerleşim yerine ve bölgelere göre KBH sıklığı)

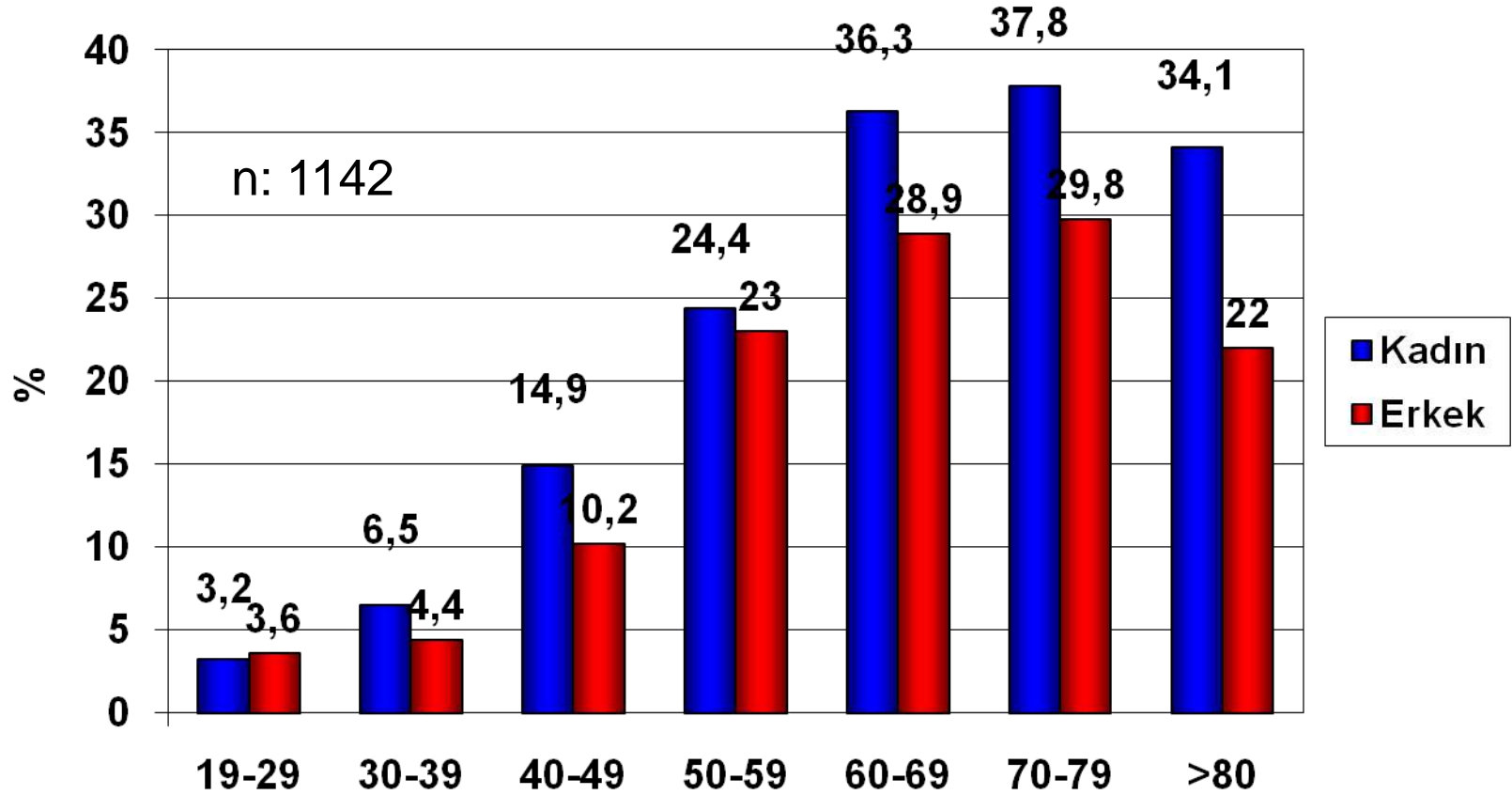


# Türkiye'de KBH ve Komorbid Hastalıklar



# Diabet Sıklığı \*

## Cins ve Yaş Gruplarına Göre



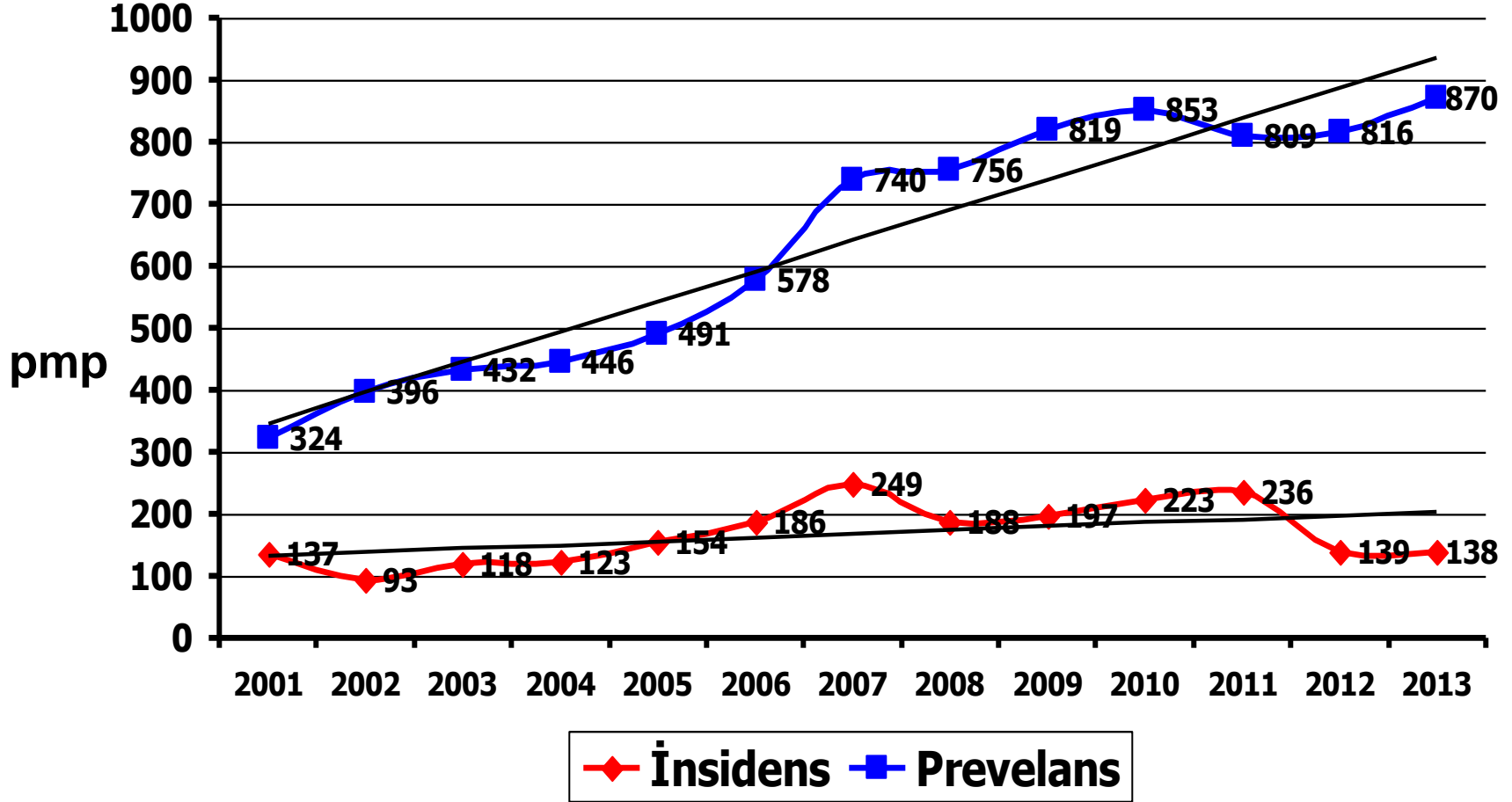
(\* :Açlık Kan Şekeri, >126 mg/dl)



# Türk toplumunda KBH için risk faktörleri: Lojistik regresyon analizi – CREDIT verileri

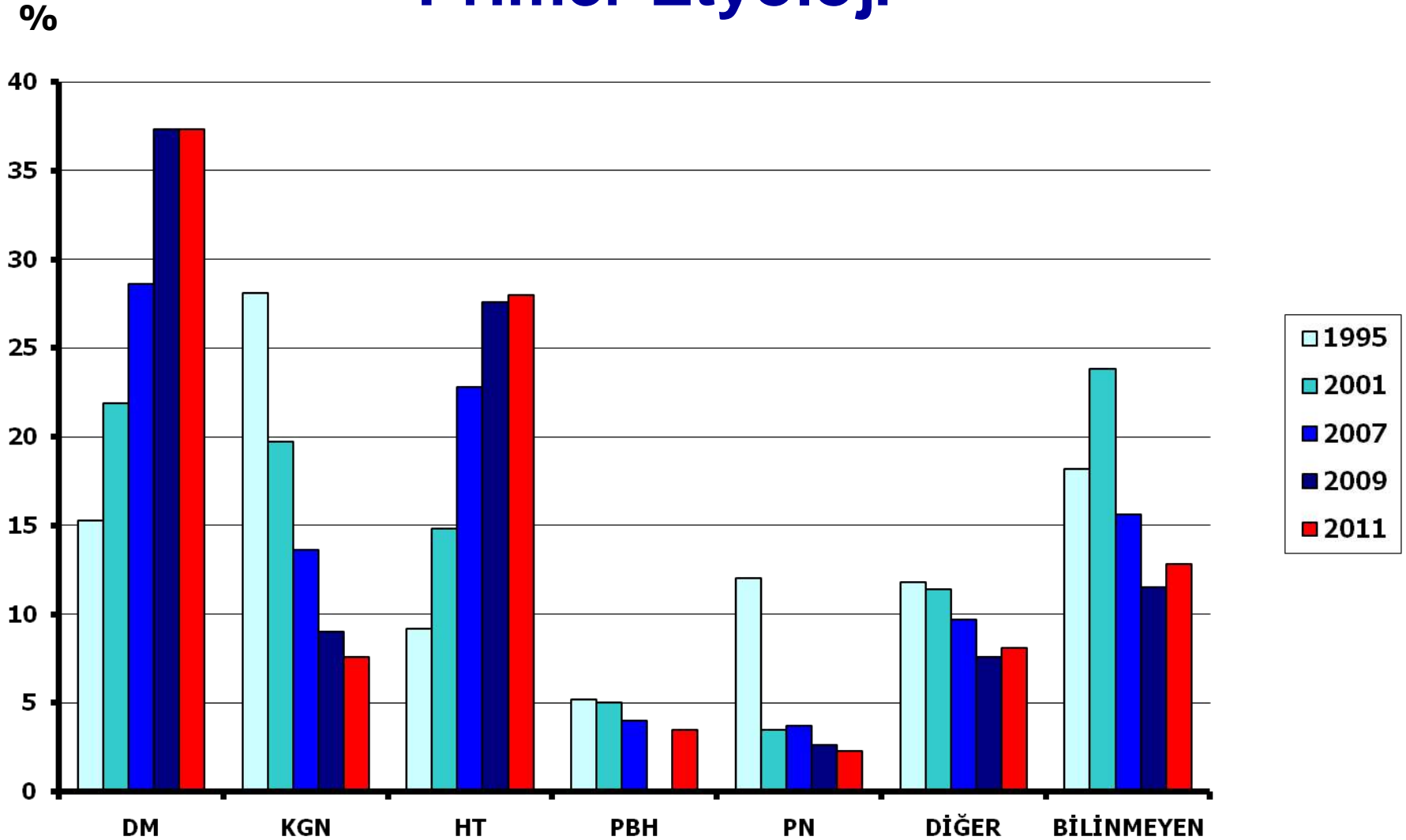
	HR	% 95 CI	P
Yaş (yıl)	1.02	1.01-1.03	<0.001
Erkek cinsiyet	0.70	0.59-0.83	<0.001
Kırsal yerleşim	1.18	1.02-1.36	0.034
Egzersiz yapma	0.83	0.71-0.96	0.011
Kalp hastalığı öyküsü	1.30	1.02-1.64	0.031
Hipertansiyon	1.38	1.17-1.62	<0.001
Diyabet	1.75	1.46-2.10	<0.001
Ürik asit düzeyi (mg/dl)	1.30	1.23-1.37	<0.001
Hemoglobin (gr/dl)	0.84	0.80-0.89	<0.001

# Türkiye' de SDBH (RRT Gerektiren) Sıklığı

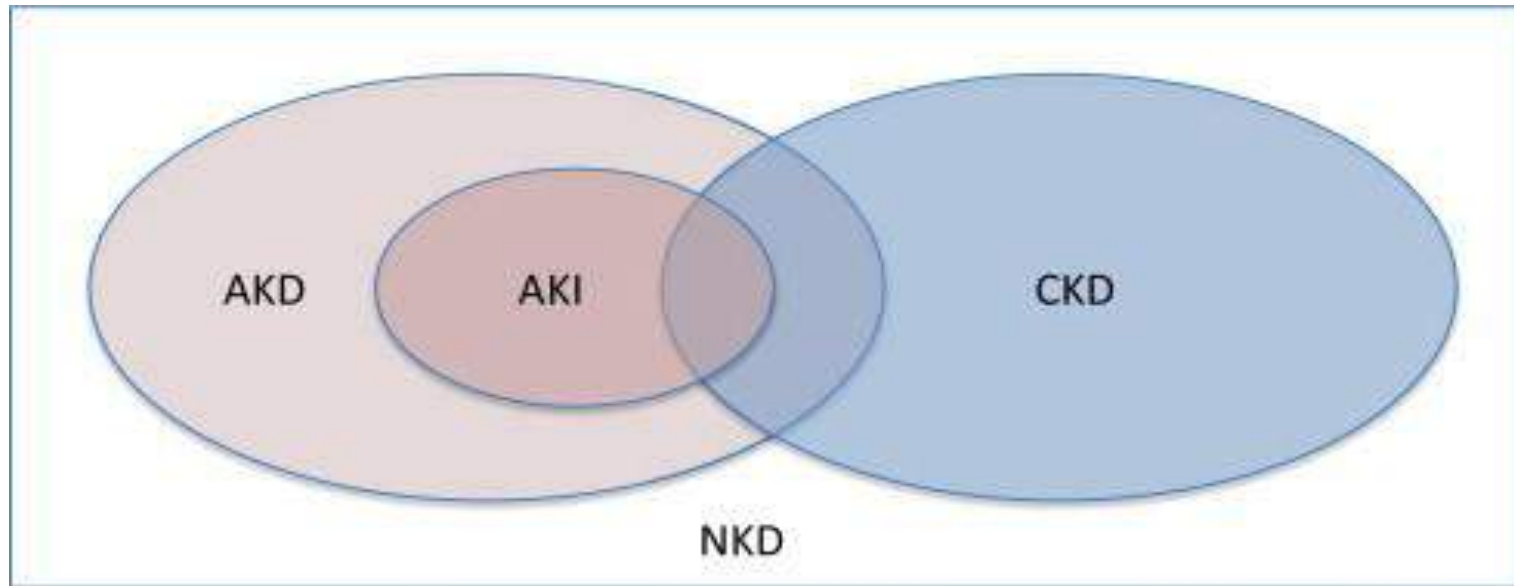


(Milyon nüfus başına;pmp)

# RRT Gerektiren SDBH nda Primer Etyoloji



# AKD (AKI) & KBH (KDIGO-2012)



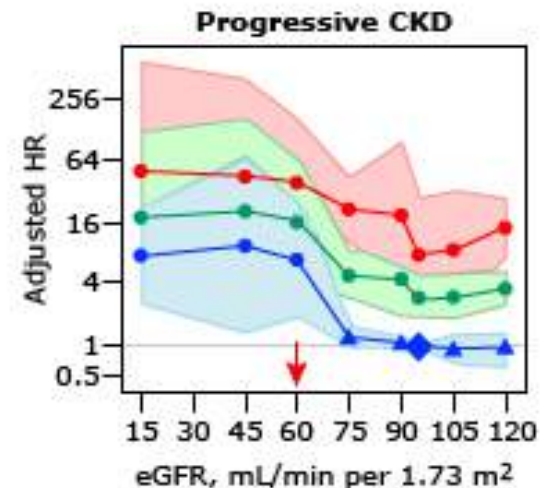
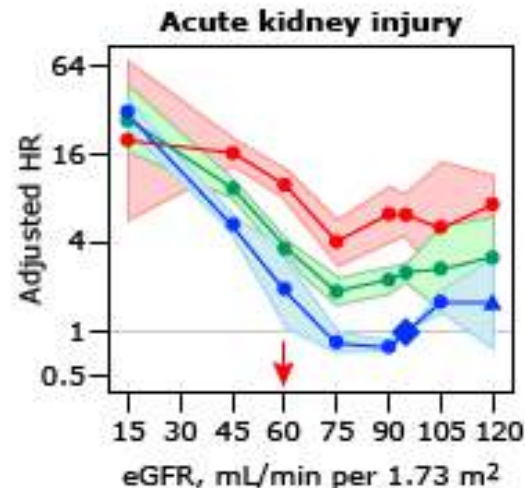
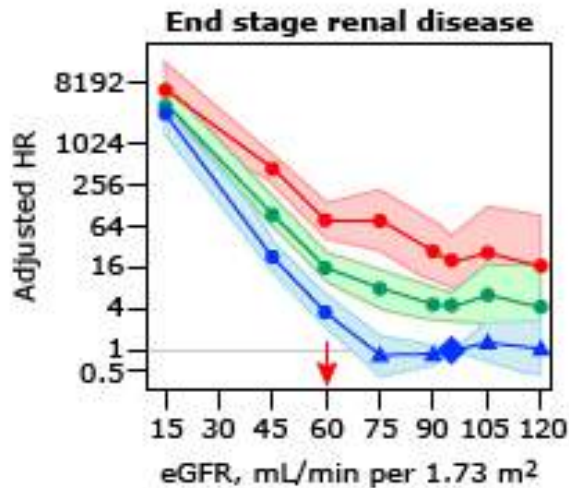
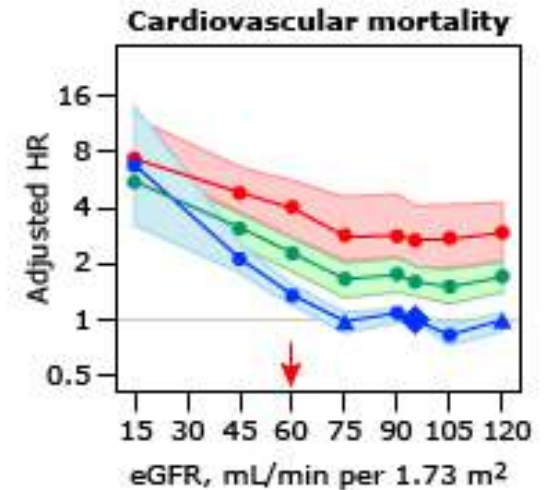
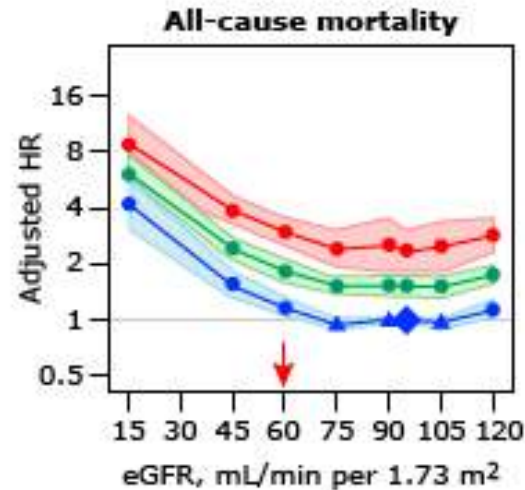
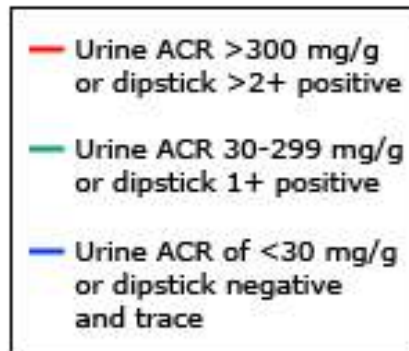
	Functional Criteria	Structural Criteria
AKI (acute kidney injury)	Increase in SCr by 50% within 7 days, or increase in SCr by 0.3 mg/dL within 2 days, or oliguria	No criteria
CKD (chronic kidney disease)	GFR <60 for >3 mo	Kidney damage for >3 mo
AKD (acute kidney diseases and disorders)	AKI, or GFR <60 for <3 mo, or decrease in GFR by ≥35% or increase in SCr by >50% for <3 mo	Kidney damage for <3 mo
NKD (no known kidney disease)	GFR ≥60, stable SCr	No kidney damage

# **Kronik Böbrek Hastalığı**

## **Tanım**

- **Üç ay veya daha uzun süren BÖBREK HASARI**  
**(GFH azalması olsun veya olmasın)**
- VE / VEYA**
- **Üç ay veya daha uzun süren GFH AZALMASI (<60 mL/dk/1.73 m<sup>2</sup>)**  
**(Böbrek hasarı olsun veya olmasın)**

# KBH & Klinik Sonuçları



# GFH ve Albuminüriye Dayanan Revize KBH Sınıflandırması (KDIGO 2012)

GFH Evreleri	GFH(ml/dk/1.73 m2)	Tanımlar
G1	≥90	Normal veya yüksek
G2	60-89	Hafif azalmış
G3a	45-59	Hafif / orta derecede azalmış
G3b	30-44	Orta / ciddi derecede azalmış
G4	15-29	Ciddi derecede azalmış
G5	<15 (veya diyaliz)	Böbrek yetmezliği (Eğer diyalizle tedavi ediliyorsa D eklenir)
Albuminüri Evreleri	AER (mg/gün)	Tanımlar
A1	<30	Normal / Yüksek normal
A2	30-300	Yüksek
A3	>300	Çok yüksek (non-nefrotik veya nefrotik olarak ayrılabilir)

# KBH & Risk Tabakalaması

				Albuminuria stages, description and range (mg/g)				
				A1		A2	A3	
				Optimal and high-normal		High	Very high and nephrotic	
				<10	10-29	30-299	300-1999	≥2000
GFR stages, description and range (mL/min per 1.73 m <sup>2</sup> )	G1	High and optimal	>105					
			90-104					
	G2	Mild	75-89					
			60-74					
	G3a	Mild-moderate	45-59					
	G3b	Moderate-severe	30-44					
	G4	Severe	15-29					
G5	Kidney failure	<15						



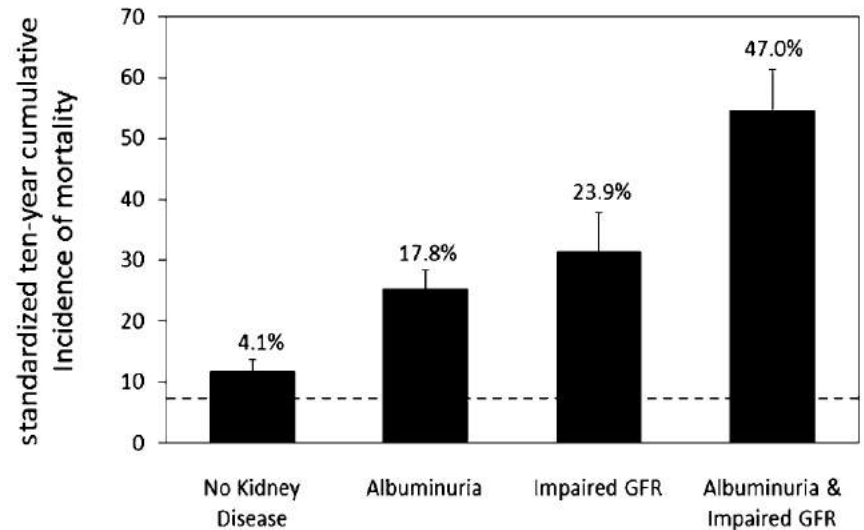
# Kidney Disease and Increased Mortality Risk in Type 2 Diabetes

Maryam Afkarian,\* Michael C. Sachs,\* Bryan Kestenbaum,\* Irl B. Hirsch,<sup>†</sup> Katherine R. Tuttle,\*<sup>‡</sup> Jonathan Himmelfarb,\* and Ian H. de Boer\*

J Am Soc Nephrol 24: 302–308, 2013

**Table 2.** Ten-year standardized all-cause and cardiovascular mortality by diabetes status

	Standardized Cumulative Incidence, % (95% CI)
All-cause mortality	
No diabetes	8.6 (7.9–9.3)
Diabetes	19.1 (15.5–22.7)
Cardiovascular mortality	
No diabetes	4.0 (3.7–4.4)
Diabetes	11.2 (8.7–13.7)
Noncardiovascular mortality	
No diabetes	6.3 (5.7–6.9)
Diabetes	13.1 (9.8–16.4)



# Diyabetin Kronik Komplikasyonları

## *Mikrovasküler Komplikasyonlar*

- **Nefropati**
- Retinopati
- Nöropati

## *Makrovasküler Komplikasyonlar*

- Koroner arter hastalığı
- Periferik arter hastalığı
- Serebrovasküler hastalık

# Diabetik Hastalardaki Böbrek Sorunları

- **Diabetik nefropati (DN)**
- Non-diabetik glomerüler hastalıklar
- Nefroskleroz
- İskemik nefropati (Aterosklerotik RAS, ateroembolik böbrek hast.)
- Akut böbrek hasarı (radyokontrast madde, nefrotoksik ajan, kardiyak sorunlara bağlı)
- İdrar yolu infeksiyonları (+papilla nekrozu)
- Sistopati (detrüsitor parezisi) ve obstrüktif üropati

# Diabetik Nefropati (DN)

- Sıklık: ~%20-40
- Tanı:
  - Persistan albüminüri (>300 mg/gün),
  - GFH ında yavaş ilerleyen azalma,
  - HT nun sıklıkla eşlik ettiği,
  - KV morbidite ve mortalitesi yüksek olan bir klinik sendromdur

# DN; Histopatoloji Bulgular

- **Glomerüler**

- Class I: GBM da kalınlaşma
- Class II: Hafif mesangial ekspansiyon
- Class III: Ağır mesangial ekspansiyon
- Class IV: Nodüler skleroz (Kimmelstiel–Wilson lezyonu)
- Class V: İleri diabetik glomeruloskleroz

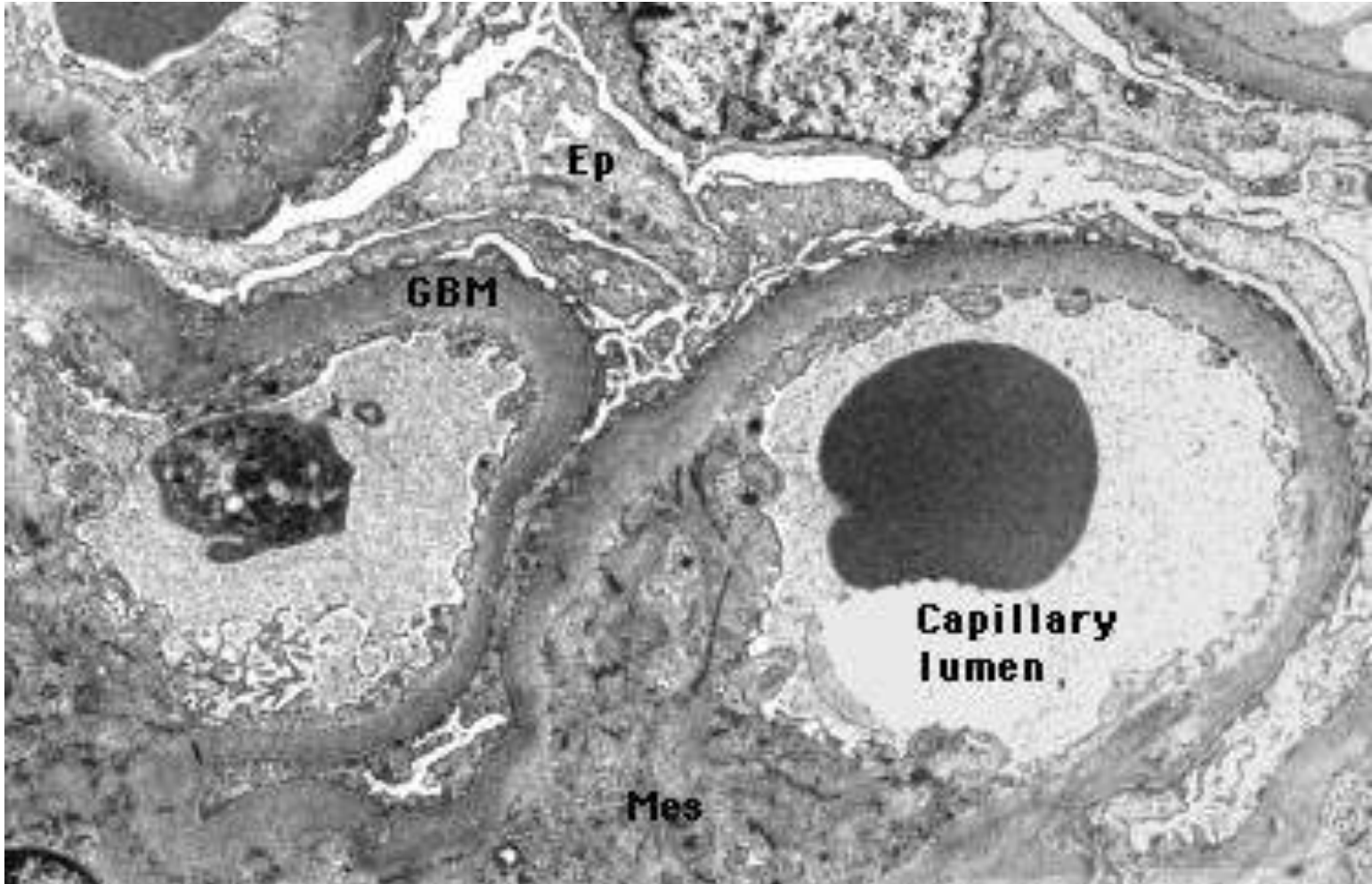
- **Vasküler**

- Arterioloskleroz, hyalin
- Ateroskleroz

- **Tubuler /Armani-Ebstein lezyonu**

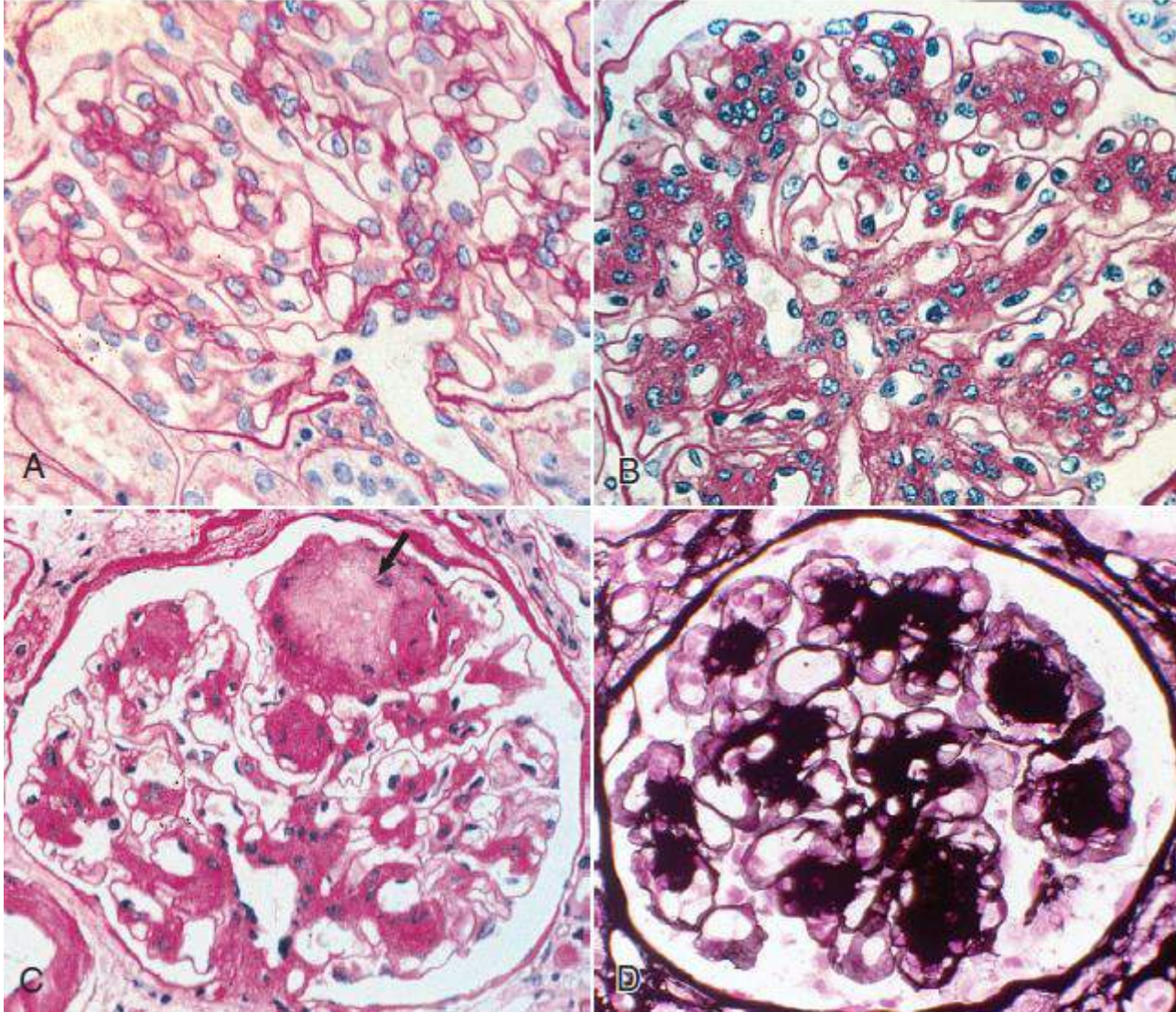
# GBM Kalınlaşması

(Class 1: K; >395 nm, E; >430 nm)

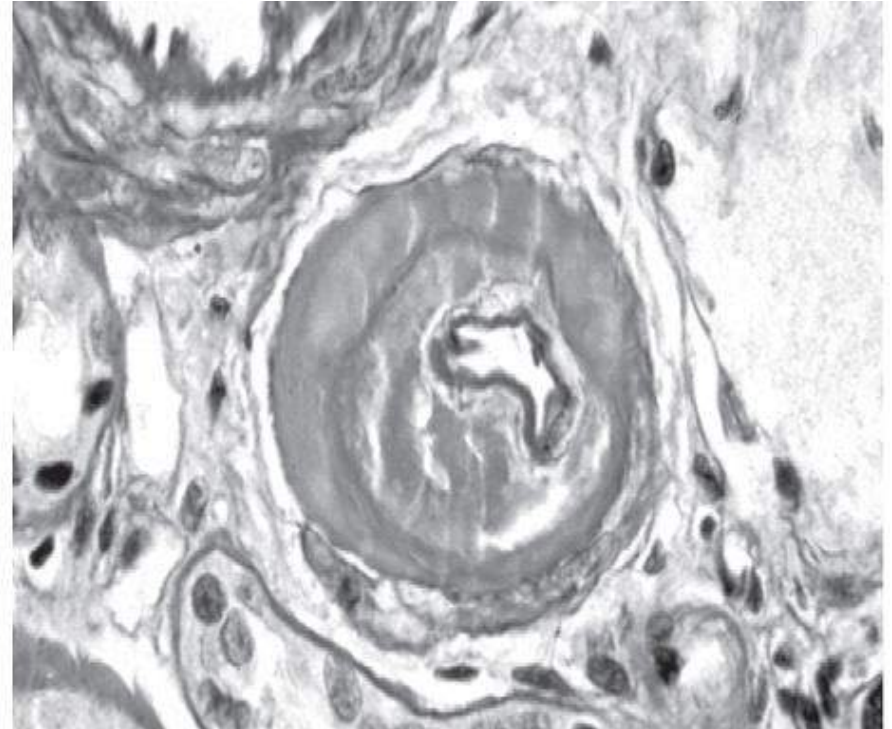
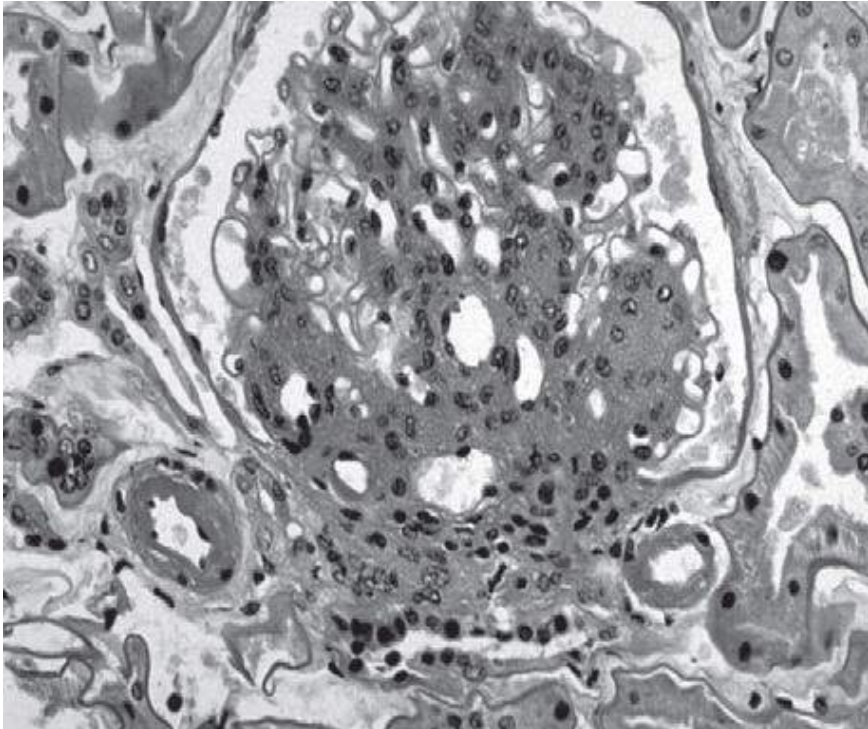




# DN de Glomerüllerdeki Yapısal Değişiklikler (Işık Mikroskopisinde)



## Tip 1 DM ta Arteriolar Hiyalinozis





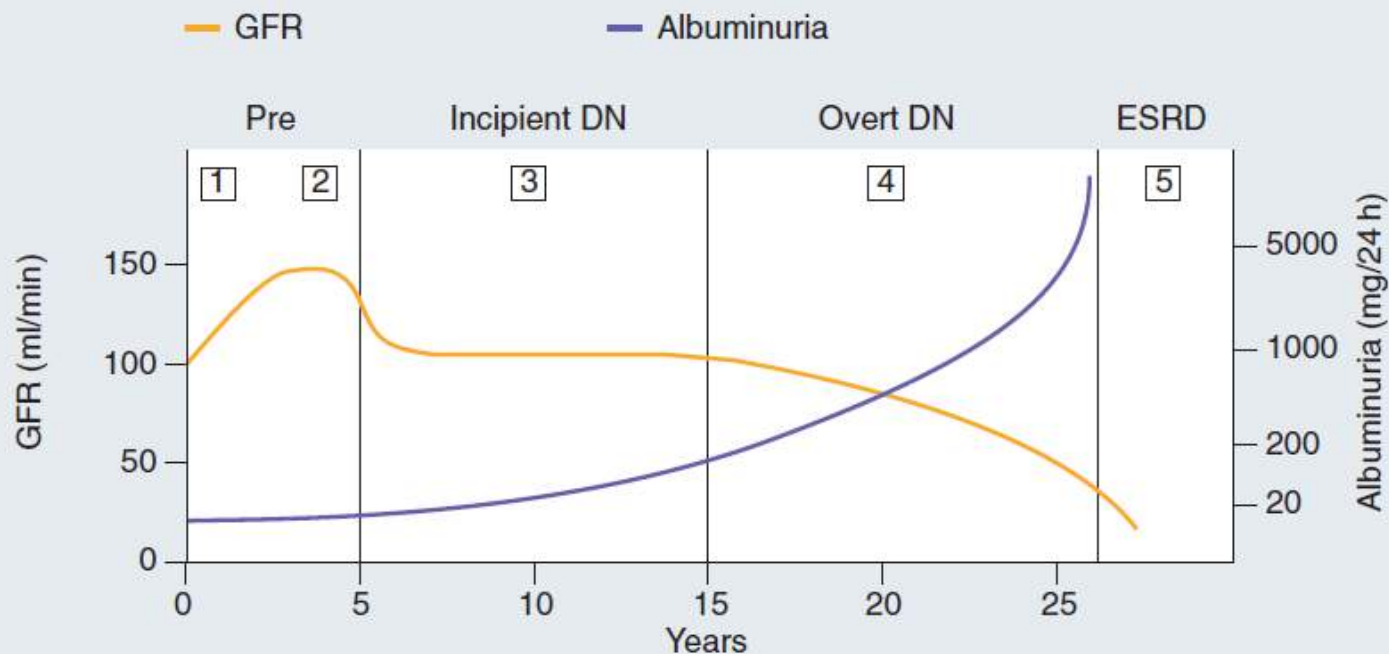
# Diabetik Nefropati Gelişimini Etkileyen Risk Faktörleri

Modifiye edilebilen	Modifiye edilemeyen
Hipertansiyon; sistemik / glomerüler	Yaş
Albuminüri / Proteinüri	Cinsiyet
Kontrol dışı hiperglisemi (Hb A <sub>1C</sub> >%7)	İrk / etnik
Dislipidemi	Genetik yatkınlık
Obezite	
Sigara	
Anemi	
Diyet (protein alımı)	

# Tip 1 Diabetin Dođal Seyri (Klinik Evreleri)

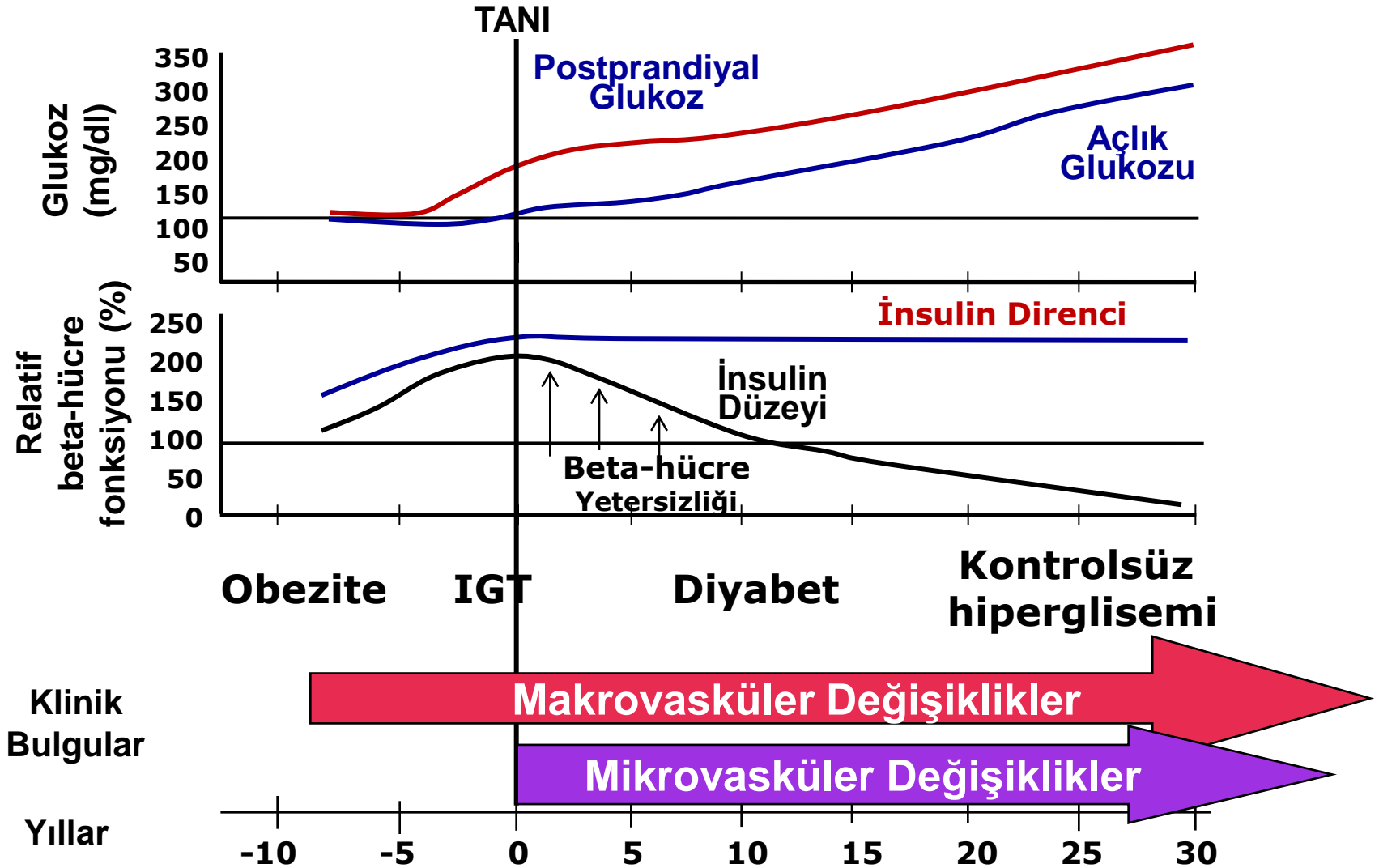
- EVRE- I: Renal hiperperfüzyon-hiperfiltrasyon-hipertrofi dönemi; tanı konulduğunda başlar
- EVRE-II: Sessiz dönem (2-5 yıl sonra); GBM kalınlaşma ve mesangial matrikste artış
- EVRE-III: Mikroalbuminüri (Incipient nefropati) (5-10 yıl)
- EVRE-IV: Makroalbuminüri (Klinik nefropati) dönemi (10-20)
- EVRE-V: Böbrek yetmezliđi dönemi (>20 yıl)

# Tip 1 Diabetin Doğal Seyri

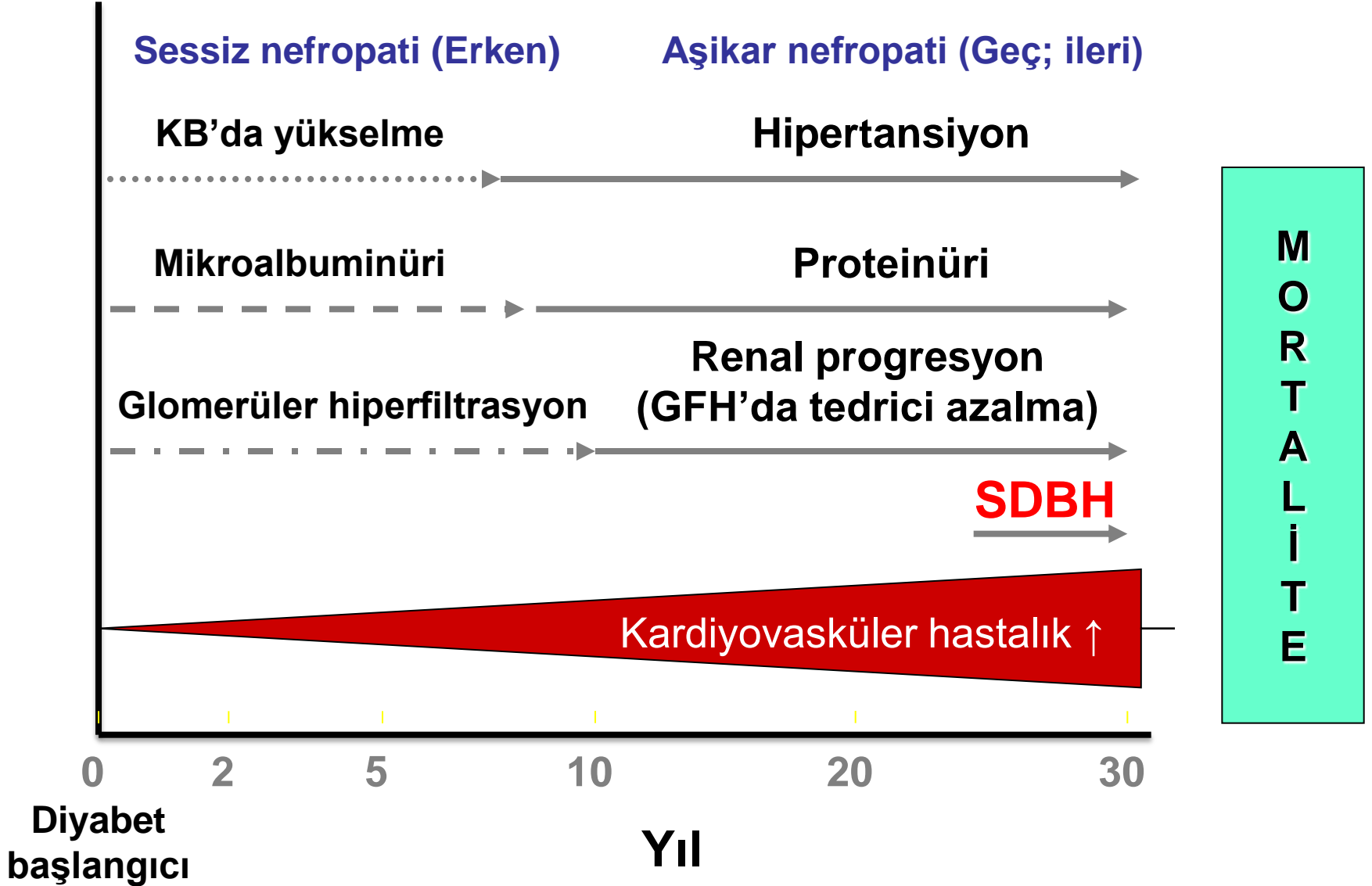


Stage	Pre	Incipient	Overt
Functional	GFR ↑ (25%–50%)	Microalbuminuria, hypertension	Proteinuria, nephrotic syndrome, GFR ↓
Structural	Renal hypertrophy	Mesangial expansion, GBM thickening, arteriolar hyalinosis	Mesangial nodules (Kimmelstiel-Wilson lesions) Tubulointerstitial fibrosis

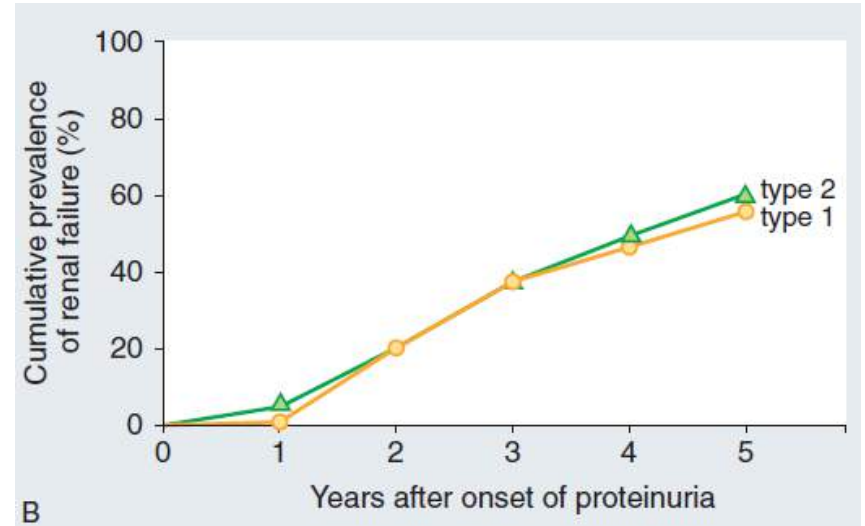
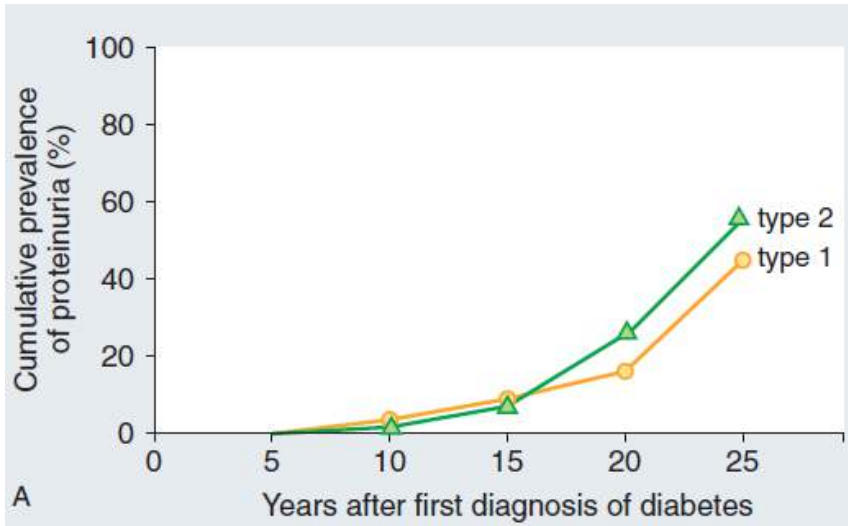
# Tip 2 Diyabetin Doğal Seyri



# Tip 2 Diyabetin Seyri

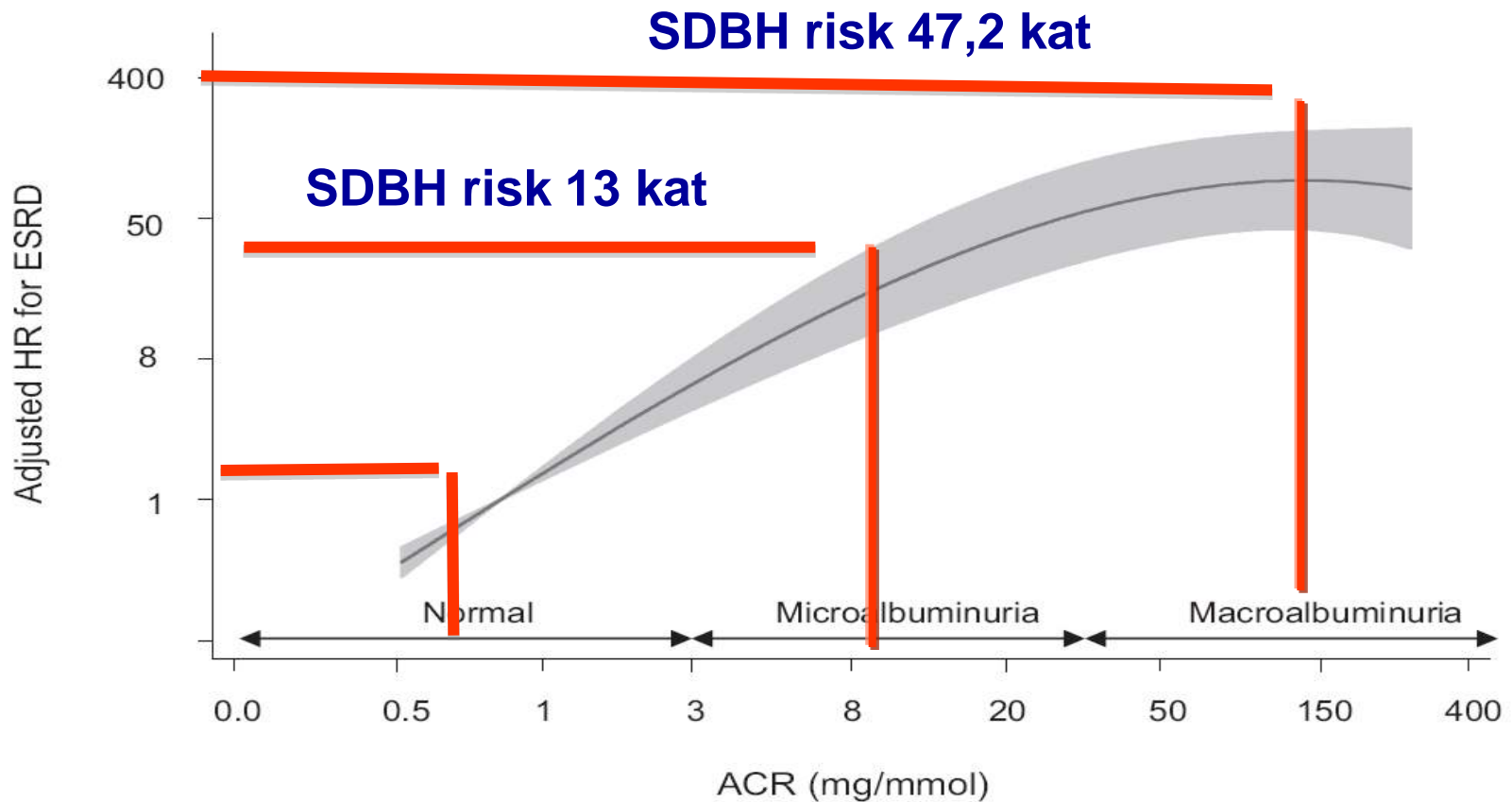


# Diabetik Hastalarda Kümülatif Proteinüri ve Böbrek Yetmezliği Gelişme Riskleri

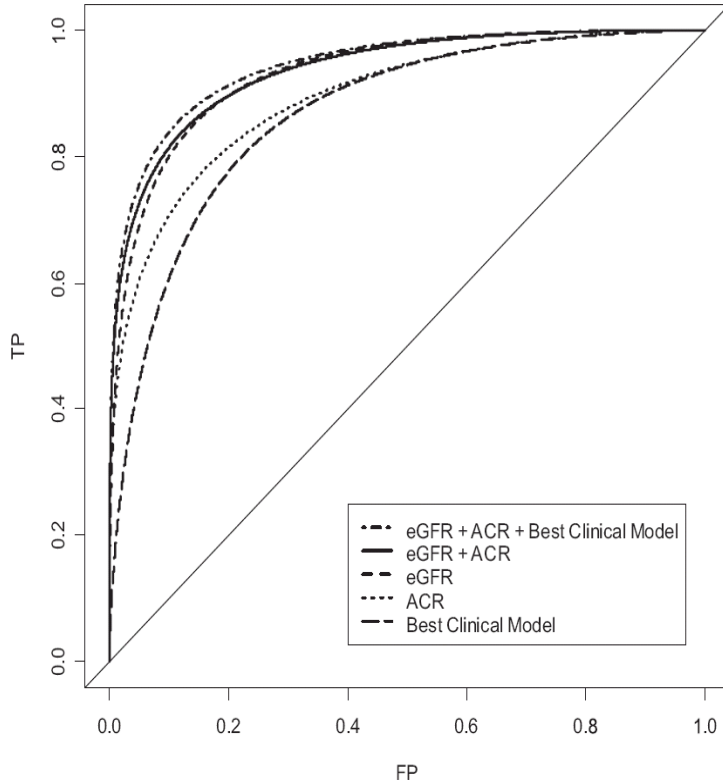


*Nephrol Dial Transplant.* 1989;4:859-863.

# Proteinüri & SDBH riski



# Albuminüri /GFH'da azalma & SDBH riski



<b>GFH, ml/dk</b>	<b>45-60</b>	<b>30-44</b>	<b>15-29</b>
<b>Risk artışı</b>	<b>6.7</b>	<b>18.8</b>	<b>65.7</b>

**Albuminüri + GFH < 60 ml/dk  
Risk artışı 570 kat**

**N=69598, 10.5 yıl**



# Diabetik Hastada Böbrek Hastalığı: Tanı /Ayırıcı Tanı

- **Klinik değerlendirme:**
  - KB ölçümü (uygun manşon, beyaz önlük HT, ortostatik değerlendirme, nokturnal HT vs)
  - Gözdibi incelemesi-Oftalmolojik değerlendirme
  - Kardiyovasküler değerlendirme
- **GFH ölçümü / tayini: SCr nin temelli formüller**
- **İdrar analizi:**
  - **Proteinüri /albuminüri varlığı, derecesi**
  - İdrar sedimenti
- **Görüntüleme yöntemleri:**
  - USG, Duplex Doppler USG, MR /CT angiografi, Eko, Koroner angio)
- **Serolojik testler:** (ANA, Anti-dsDNA, ANCA, C3-C4 vs)
- **Böbrek biyopsisi: Işık M, IFM, EM**

# Diabetik Nefropati (DN) (Klinik Tanı)

- Hastalık süresi >5-10 yıl
- Persistan albüminüri
  - Mikroalbuminüri (30-299 mg/gün) (>5 yıl)
  - Makroalbuminüri (>300 mg/gün) (>10yıl)
- Yavaş ilerleyen GFH azalması
- HT varlığı (Tip 2 > Tip 1)
- Retinopati: Tip 1;~%100, Tip 2;%50-60
- İdrar sedimenti inaktiftir, mikroskopik hematüri bulunabilir, makroskopik hematüri yoktur, eritrosit silendiri yoktur
- USG de, Bb normal veya büyüktür
- Çoğu olguda tanı için BBx gerekmez

# Non-proteinürik Diabetik Nefropati

- DN nin klasik klinik paradigması normoalbuminüriden proteinüriye oradan da böbrek yetmezliğine giden bir yol izler
- DN nin klasik klinik paradigması BF azalması olan her diabetik hastada görülmeyebilir
- DN nin bir alt grubunda proteinüri ya yoktur ya da minimaldir
- Olası mekanizmalar:
  - RAS ni bloke eden ilaçların kullanımı
  - Tam gerilemeyen AKI atakları
  - Genetik predizpozisyon
  - Diğer non-diabetik böbrek hastalıklar

Diabetes Care 2007;30(8):2034–9  
JAMA 2003;289(24):3273–7  
Cardiorenal Med 2012;2(1):1–10

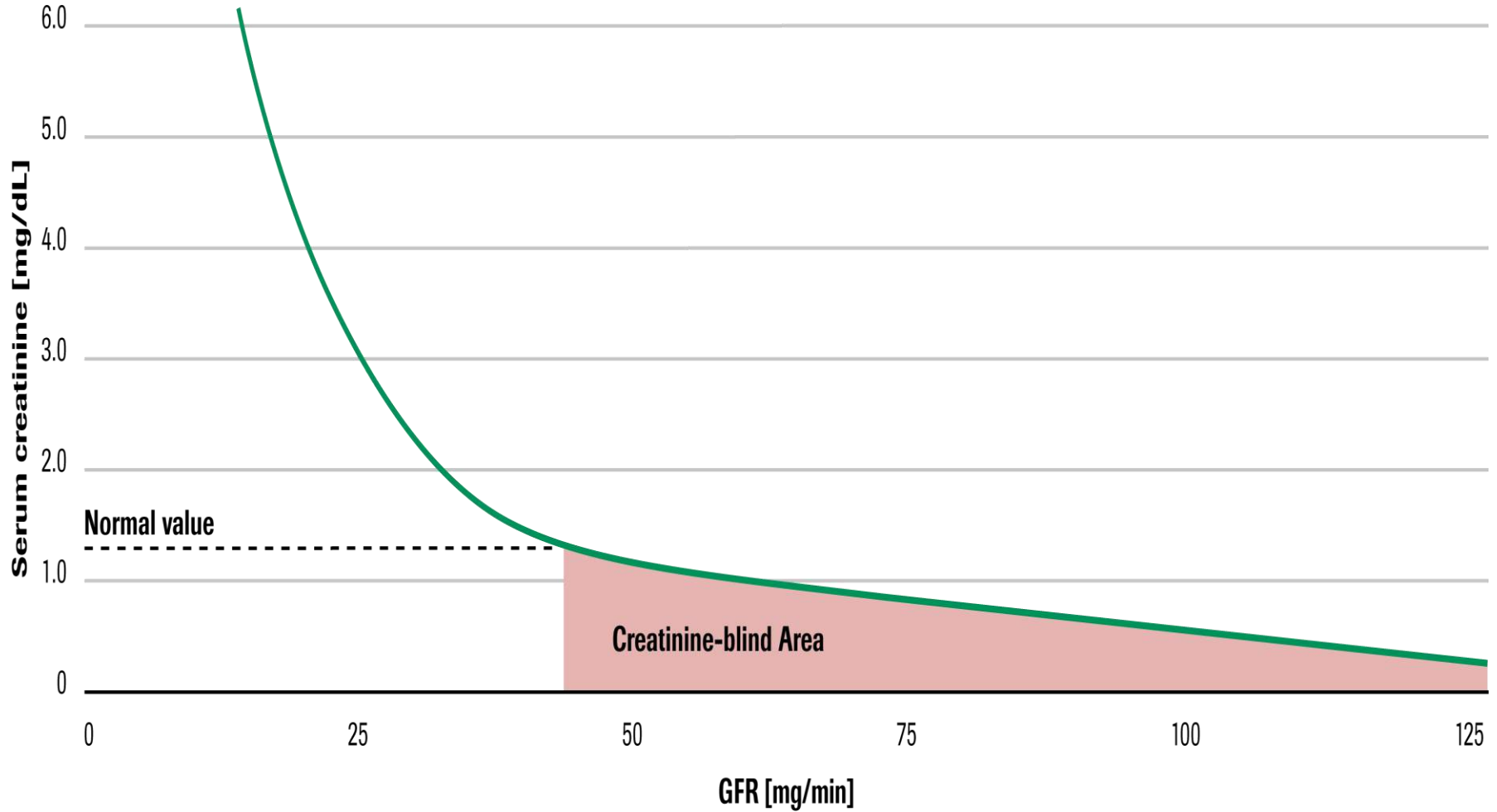
# Non-diabetik Böbrek Hastalıklar

- Beş yıldan kısa öyküsü olan Tip 1 DM olgularında proteinüri gelişmesi
- Mikroalbuminüri aşamasından geçmeden masif proteinüri gelişmesi
- Albuminüri yokluğunda böbrek fonksiyon bozukluğu
- Akut ve/veya hızlı gelişen böbrek fonksiyon bozukluğu
- Makroskopik hematüri varlığı
- Aktif idrar sedimenti ve hücresel silendirlerin bulunması
- Tip 1 diabetiklerde retinopati veya nöropatinin bulunmaması. Ancak Tip 2 diabetiklerde bu ilişki o kadar kuvvetli değildir
- Diğer sistemik hastalıklara (SLE, MM vs) ilişkin belirti ve bulguların varlığı
- RAS blokörü verilmesinden sonraki GFH ında >%20-30 azalma olması

# Glomerüler Filtrasyon Hızı (GFH)

- Serum kreatinin düzeyi (SCr) ve  $1/SCr$
- Endojen kreatinin klirensi (CCr)
- Simetidimli kreatinin klirensi
- $(\text{Üre klirensi} + \text{Kreatinin klirensi}) / 2$
- Sistatin C tayini
- İnülin klirensi (Cin)
- Radyonüklid endikatörlü klirensi (Iotalamat, EDTA, DTPA)
- Radyokontrast madde klirensi (İoheksol)
- Hesaplanmış kreatinin klirensi
  - Cockroft-Gault eşitliği
  - MDRD formülü (GFH)
  - CKD-EPI formülü

# SCr & GFH İlişkisi



# GFH Hesaplamaya Yönelik Kreatinin Temelli Formüller

## Erişkin

- *Cockcroft Gault eşitliği, 1976*
- *MDRD eşitliği, 1999*
- *CKD-EPI eşitliği, 2009*

## Çocuk

- *Schwartz eşitliği, 1976*

# Böbrek Hasarı Göstergeleri

- **Persistan mikroalbuminüri**
- **Persistan albuminüri / proteinüri**
- Persistan hematüri
- Görüntüleme yöntemlerinde (USG vs) böbreklerde yapısal anormallik varlığı
- Böbrek biyopsisinde patolojik bulgular (GN vs)



# İdrarda Protein Araştırması

- Albümin dipstick leri
- 24 saatlik protein atılımı ölçümü
- Random (spot) idrar örneğinde
  - Protein/kreatinin oranı (PCR)
  - **Albümin/kreatinin oranı (ACR)**

# Mikroalbuminüri- Karıştırıcı Faktörler

- Egzersiz
- İnfeksiyon; genel, üriner
- Ateş
- Belirgin hiperglisemi
- Gebelik
- Menstruasyon
- Kalp yetmezliği

# Böbrek Zedelenmesi Göstergesi olarak Albüminüri

	<b>AER (mg/gün)</b>	<b>ACR (mg/gram kreatinin)</b>
<b>İdrar toplama şekli</b>	<b>24 saat</b>	<b>Spot idrar (tercihan sabah)</b>
<b>Normoalbuminüri (A1)</b>	<b>&lt;30</b>	<b>&lt;30</b>
<b>Mikroalbuminüri (A2)</b>	<b>30-300</b>	<b>30-300</b>
<b>Makroalbuminüri (A3)</b>	<b>&gt; 300</b>	<b>&gt; 300</b>

# Proteinürinin Genel Mortalite ve Böbrek Yetmezliğine Etkileri

**All-cause mortality**

	ACR <10	ACR 10-29	ACR 30-299	ACR ≥300
eGFR >105	1.1	1.5	2.2	5.0
eGFR 90-105	Ref	1.4	1.5	3.1
eGFR 75-90	1.0	1.3	1.7	2.3
eGFR 60-75	1.0	1.4	1.8	2.7
eGFR 45-60	1.3	1.7	2.2	3.6
eGFR 30-45	1.9	2.3	3.3	4.9
eGFR 15-30	5.3	3.6	4.7	6.6

**Kidney failure (ESRD)**

	ACR <10	ACR 10-29	ACR 30-299	ACR ≥300
eGFR >105	Ref	Ref	7.8	18
eGFR 90-105	Ref	Ref	11	20
eGFR 75-90	Ref	Ref	3.8	48
eGFR 60-75	Ref	Ref	7.4	67
eGFR 45-60	5.2	22	40	147
eGFR 30-45	56	74	294	763
eGFR 15-30	433	1044	1056	2286

# Diabetiklerde Mikroalbuminüri / Proteinüri Taraması

- Tip I DM; 5. yıldan sonra yılda bir kez
- Tip II DM; tanı konulduktan sonra yılda bir kez

# **İdrar Sedimenti Analizi**

## Clinical significance of urinary casts

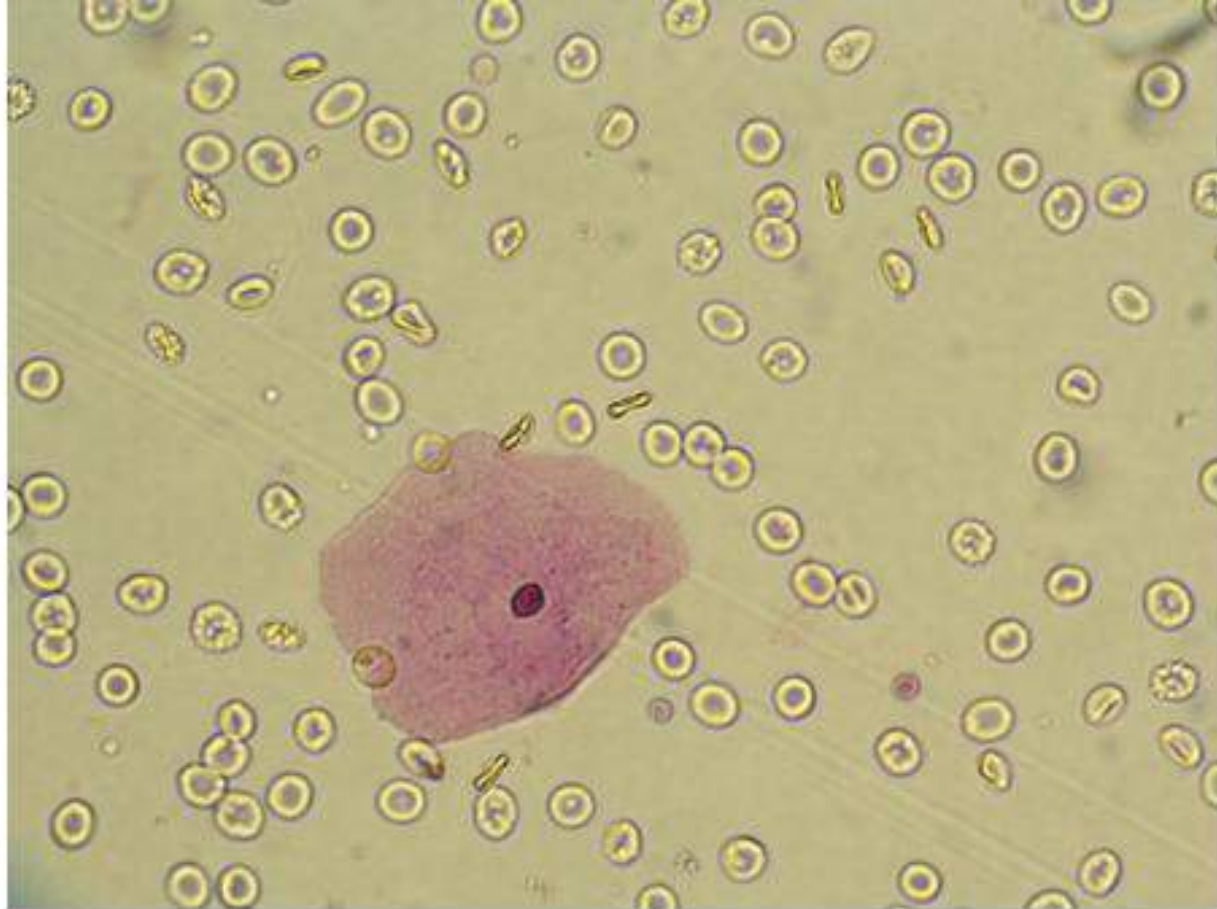
Cast	Main clinical Associations
Hyaline	Normal subject Renal disease
Granular	Renal disease
Waxy	Renal insufficiency Rapidly progressive Glomerulonephritis
Fatty	Marked proteinuria Nephrotic syndrome
Erythrocyte	Glomerular bleeding Proliferative/necrotizing glomerulonephritis
Hemoglobin	Glomerular bleeding Proliferative/necrotizing glomerulonephritis Hemoglobinuria
Leukocyte	Acute pyelonephritis Acute interstitial nephritis Proliferative glomerulonephritis
Epithelial	Acute tubular necrosis Acute interstitial nephritis Glomerulonephritis
Myoglobin	Rhabdomyolysis

# Glomerüler / Non-glomerüler Hematüri Ayırımı

<b>ÖZELLİK</b>	<b>GLOMERÜLER</b>	<b>EKSTRA- GLOMERÜLER</b>
<b>RENK</b>	<b>Kırmızı / Kahverengi</b>	<b>Kırmızı / Pembe</b>
<b>PIHTI</b>	<b>Yok</b>	<b>Olabilir</b>
<b>ERİTROSİT MORFOLOJİSİ</b>	<b>Anormal Dismorfik (%10-80,ort %40;akantosit &gt;%5)</b>	<b>Normal İzomorfik</b>
<b>ERİTROSİT SİLENDİRİ</b>	<b>Olabilir</b>	<b>Yok</b>
<b>PROTEİNÜRİ</b>	<b>&gt;500 mg/gün</b>	<b>&lt;500 mg/gün</b>



# Monomorfik (İzomorfik) eritrositler



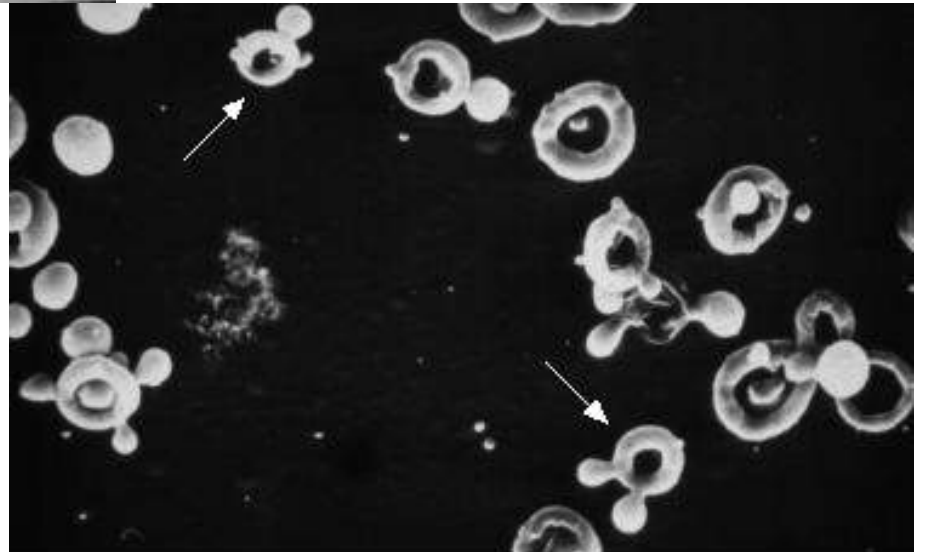
# Dismorfik Eritrositler

(Faz Kontrast Mikroskopisi)

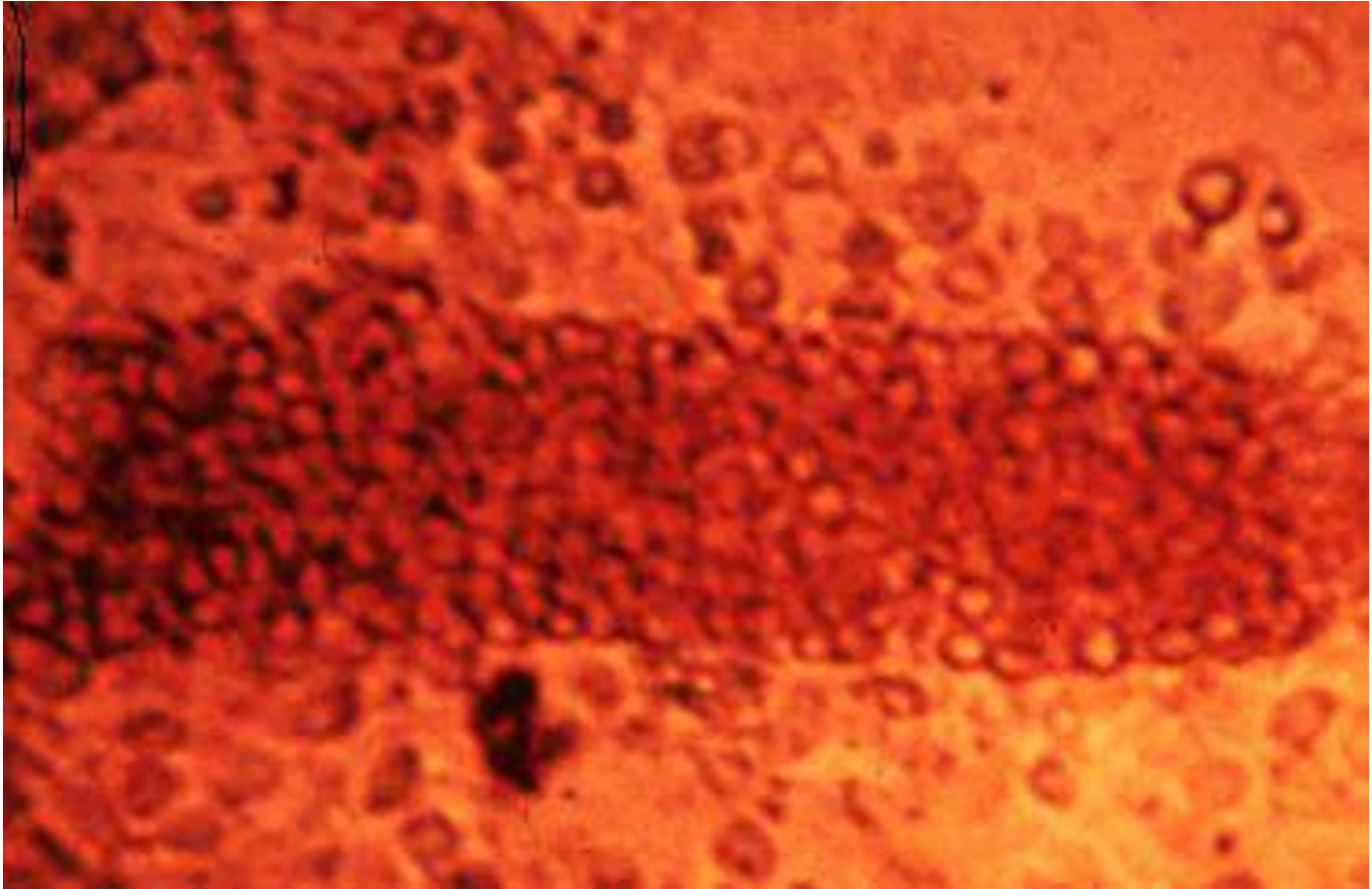


akantosit

(Scanning Elektronmikroskopisi)



# Eritrosit Silendiri



**Diabetes proteinuria**

Exclude urinary tract infection  
Urine microscopy: red cells, white-cell casts?  
Quantitate proteinuria  
Renal ultrasonography  
Serology if glomerulonephritis suspected  
ANCA, DNA antibodies, C3, C4

**Typical diabetic nephropathy**

Type 1 diabetes for >10 years  
Retinopathy  
Previous microalbuminuria  
No macroscopic hematuria  
No red cell casts  
Enlarged kidneys on ultrasound

No renal biopsy

**Atypical proteinuria**

Type 1 diabetes for <10 years  
No retinopathy  
Nephrotic range proteinuria  
without progression through  
microalbuminuria  
Macroscopic hematuria  
Red cell casts

Renal biopsy

**Atypical**

Azotemia with proteinuria <1 g/day  
Papillary necrosis (pyuria,  
hematuria, scarring)  
Tuberculosis (pyuria, hematuria)  
Renovascular disease (other  
occlusive vascular disease)

No renal biopsy

# Glomerüler Hastalıklara Bağlı Major Klinik Sendromlar

- Akut glomerülonefrit (Akut nefritik sendrom, AGN)
- Hızlı ilerleyen glomerülonefrit (RPGN)
- Makroskopik hematüri
- Nefrotik sendrom (NS)
- Asemptomatik persistan idrar anormallikleri
- Kronik glomerülonefrit (KGN)



# Clinical Presentations of Glomerular Disease

## Asymptomatic

Proteinuria 150 mg to 3 g per day  
Hematuria >2 red blood cells  
per high-power field in spun urine  
or  $>10 \times 10^6$  cells/liter  
(red blood cells usually dysmorphic)

## Macroscopic hematuria

Brown/red painless hematuria  
(no clots); typically coincides with  
intercurrent infection  
Asymptomatic hematuria  $\pm$  proteinuria  
between attacks

## Nephrotic syndrome

Proteinuria: adult  $>3.5$  g/day;  
child  $>40$  mg/h per  $m^2$   
Hypoalbuminemia  $<3.5$  g/dl  
Edema  
Hypercholesterolemia  
Lipiduria

## Nephritic syndrome

Oliguria  
Hematuria: red cell casts  
Proteinuria: usually  $<3$  g/day  
Edema  
Hypertension  
Abrupt onset, usually  
self-limiting

## Rapidly progressive glomerulonephritis

Renal failure over days/weeks  
Proteinuria: usually  $<3$  g/day  
Hematuria: red cell casts  
Blood pressure often normal  
May have other features of vasculitis

## Chronic glomerulonephritis

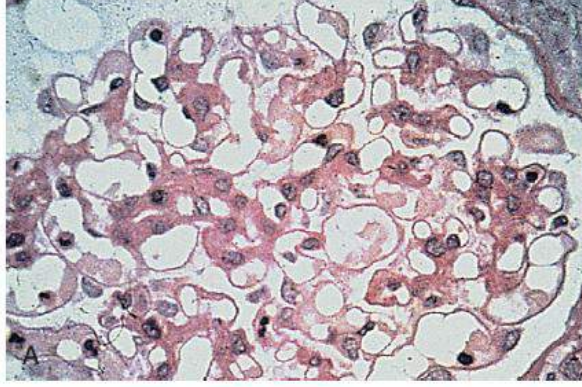
Hypertension  
Renal insufficiency  
Proteinuria often  $>3$  g/day  
Shrunken smooth kidneys

# Glomerüler Hastalıklar (Klinik Sendromlar)

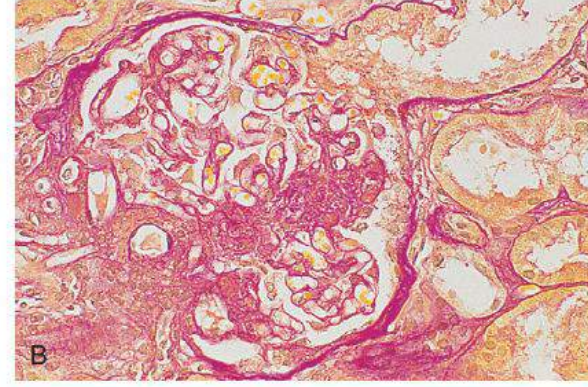
<u><i>Glomerüler Hastalık</i></u>	<u><i>Nefrotik</i></u>	<u><i>Nefritik</i></u>
• Minimal Değ. Glomerülopati	++++	-
• Membranöz Glomerülopati	++++	+
• Diabetik Glomerüloskleroz	++++	+
• Amiloid Nefropatisi	++++	+
• Fokal Seg. Glomerüloskleroz	+++	++
• Fibriler Glomerülonefrit	+++	++
• Mezangioproliferatif GN	++	++
• Membranoproliferatif GN	++	+++
• Akut Diffüz Proliferatif GN	+	++++
• Kresentik GN	+	++++

# Glomerüler Hastalıklarda Patoloji: Işık Mikroskopisi

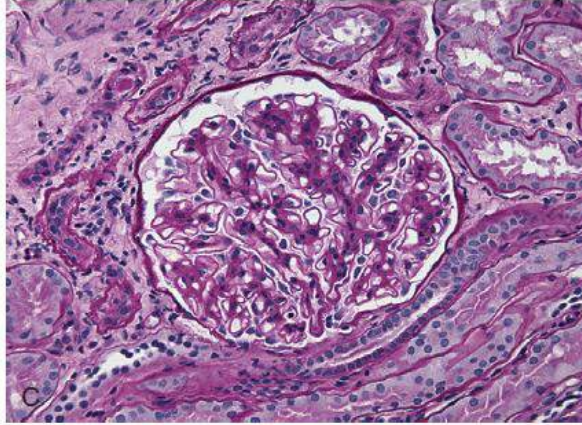
**Minimal  
Değişiklik  
Hastalığı**



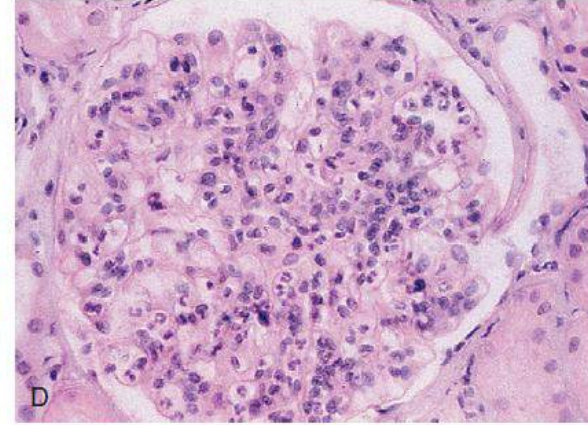
**FSGS**



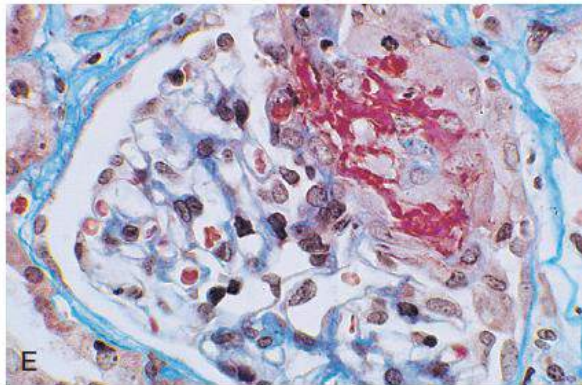
**IgA N**



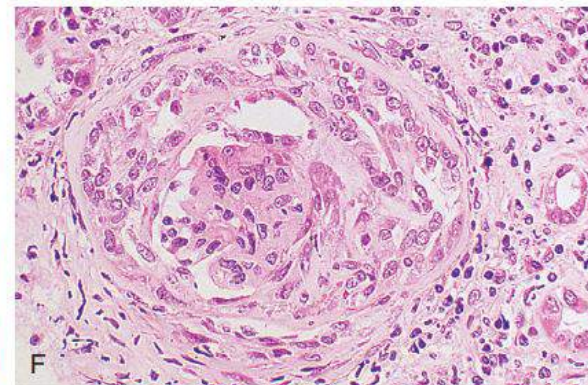
**APSGN**



**Renal  
Vaskülit**

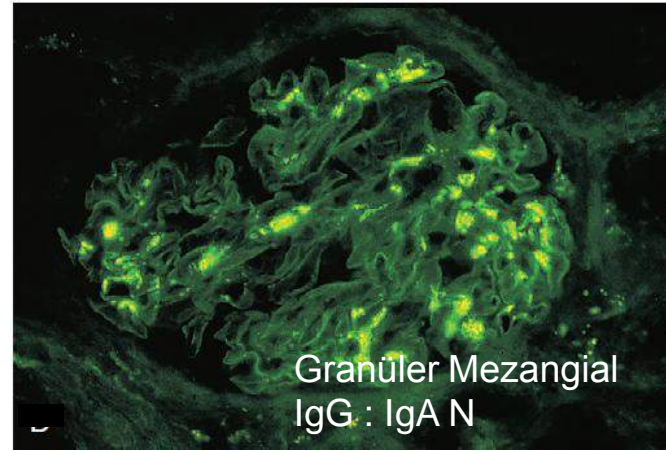
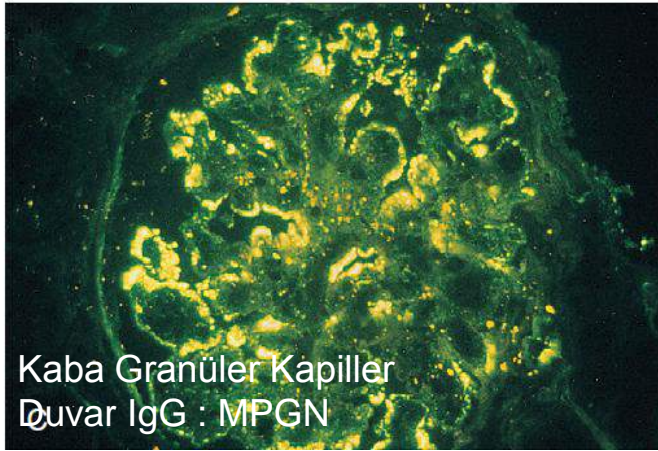
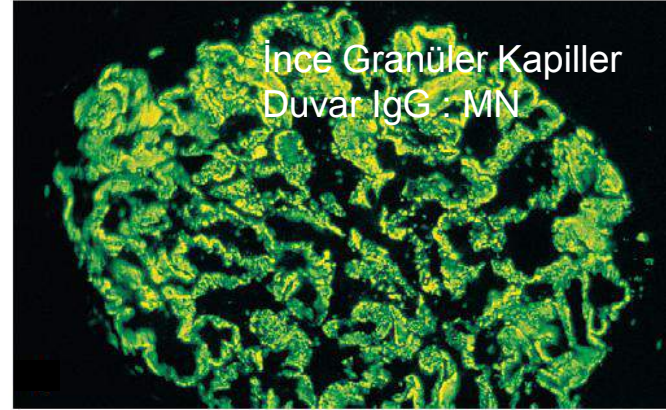
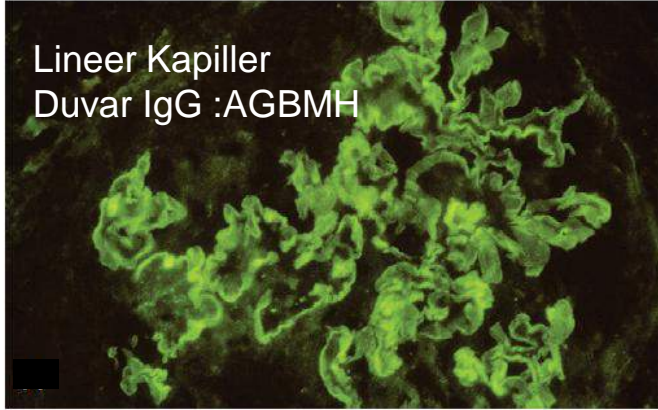


**AGBMH  
Kresentik GN**



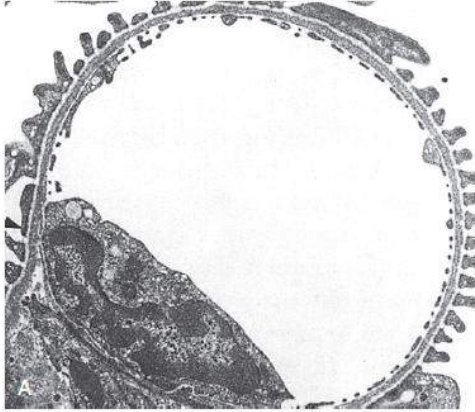


# Glomerüler Hastalıklarda Patoloji: İmmunfloresan Mikroskopisi



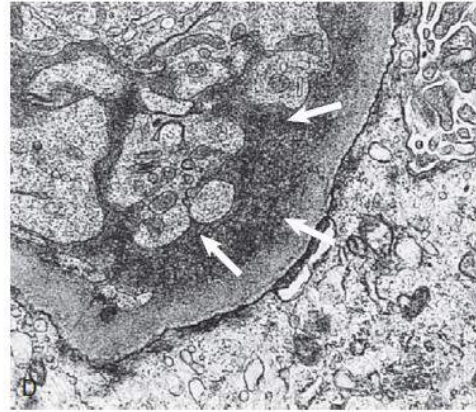
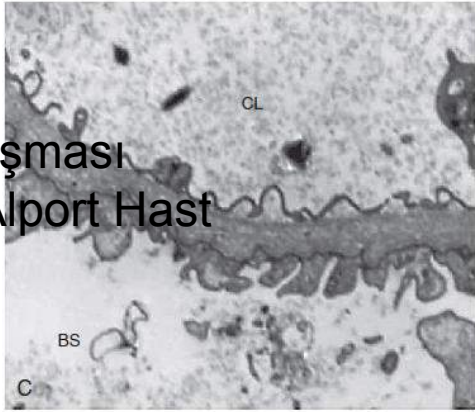
# Glomerüler Hastalıklarda Patoloji: Elektron Mikroskopisi

Normal  
Glomerülüs



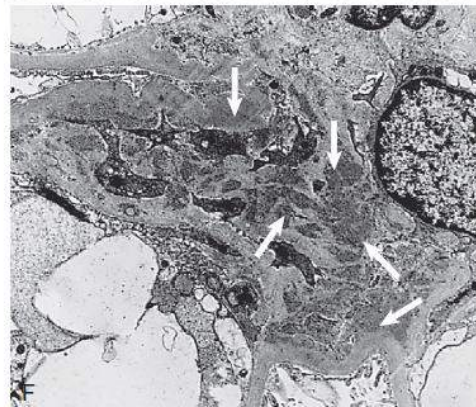
Ayaksı Uzantı  
Füzyonu:MDH

GBM kalınlaşması  
Ayrışması: Alport Hast



Subendotelial DD  
Birikimi: MPGN

Subepitelial DD  
Birikimi: MN

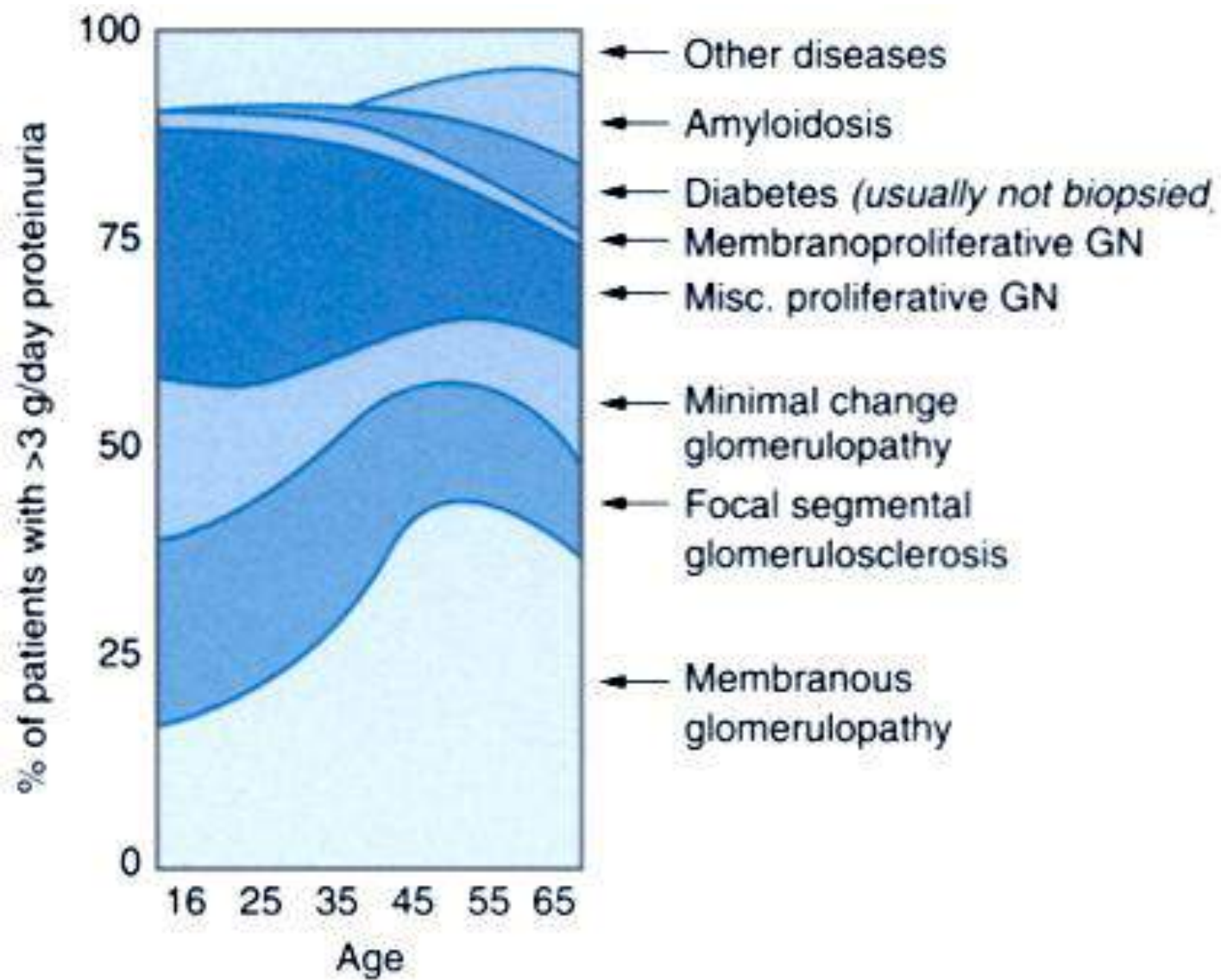


Mezangial DD  
Birikimi: IgA N



# Diabet Dışı Mekanizmalarla Gelişen Glomerüler Hastalıklar

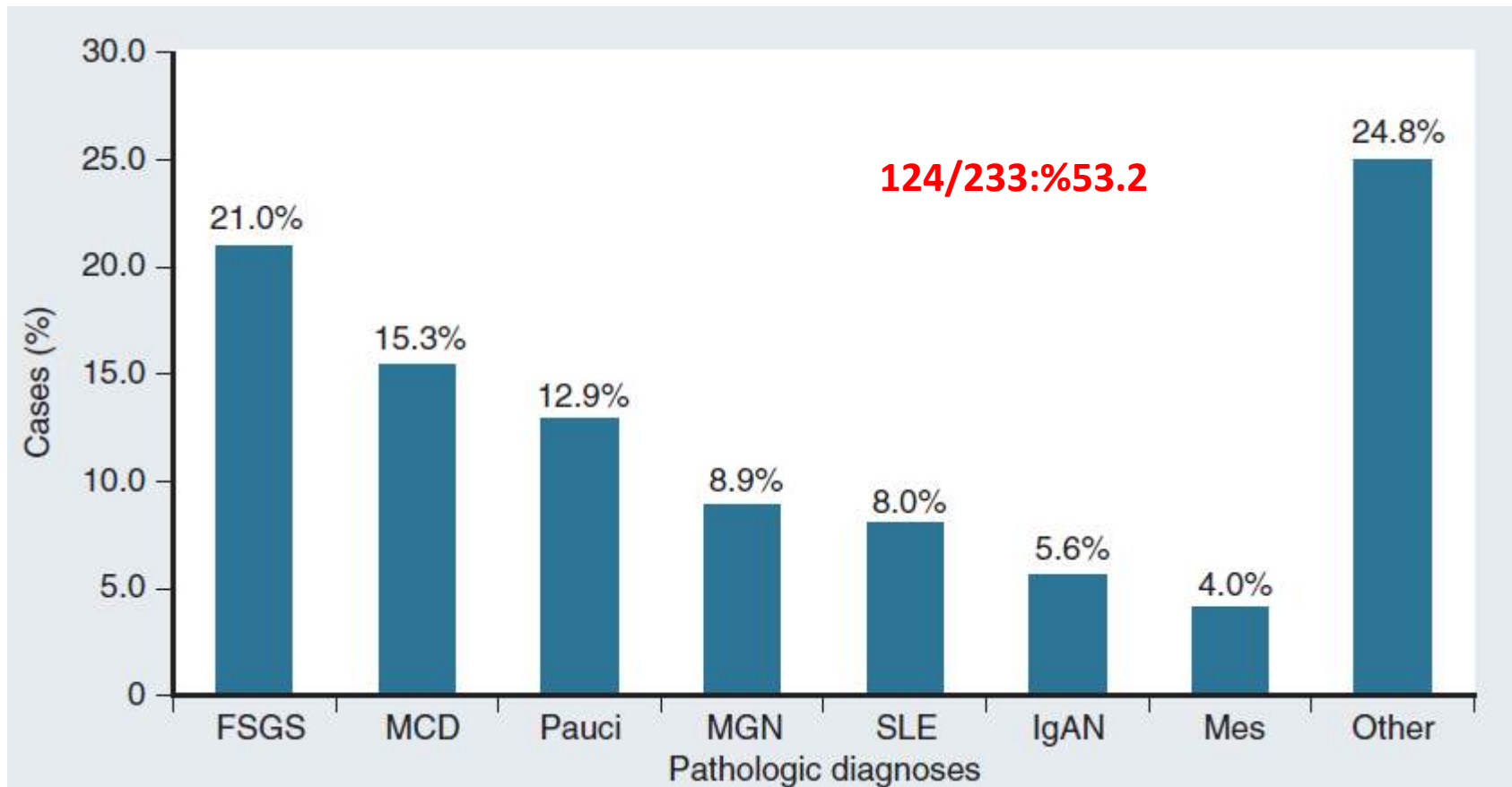
- Membranöz nefropati,
- Minimal değişiklik hastalığı,
- IgA nefropatisi,
- Fokal segmental glomeruloskleroz,
- Henoch-Schönlein purpurası,
- İnce bazal membran hastalığı,
- Proliferatif glomerulonefrit,
- *Collapsing* glomerulopati
- *Pauci-immune* kresentik glomerulonefritler



# Prevalence of nondiabetic renal disease in diabetic patients

Pham TT, Sim JJ, Kujubu DA, et al.

*Am J Nephrol.* 2007;27:322-328



# The Modern Spectrum of Renal Biopsy Findings in Patients with Diabetes

Shree G. Sharma,\* Andrew S. Bomback,† Jai Radhakrishnan et al.

Clin J Am Soc Nephrol 8: 1718–1724, 2013

- Diabetik hasta : 620
- Erkek hasta: 371
- Yaş, median(aralık): 62 (52-69)
- DM öyküsü: Ort 10 yıl
- İleri Evre KBH (4-5): %52
- **Patolojik tanı:**
  - DN n:227 (%37)
  - NDRD n:220 (%36)
  - DN+NDRD n:164 (%27)

# The Modern Spectrum of Renal Biopsy Findings in Patients with Diabetes

Shree G. Sharma,\* Andrew S. Bomback,† Jai Radhakrishnan,† et al and Vivette D. D'Agati‡

Clin J Am Soc Nephrol 8: 1718–1724, 2013

## NDRD Grubundaki hastalarda

- FSGS %22
- Nefroskleroz %18
- ATN %17
- Pauciimmün GN %13
- IgA N %11
- MN %8

# Pauci-immune Crescentic Glomerulonephritis Superimposed on Diabetic Glomerulosclerosis

Samih H. Nasr,\* Vivette D. D'Agati,\* Samar M. Said,\* Michael B. Stokes,\* Gerald B. Appel,<sup>†</sup> Anthony M. Valeri,<sup>†</sup> and Glen S. Markowitz\*

*\*Department of Pathology and <sup>†</sup>Department of Medicine, Division of Nephrology, Columbia University, College of Physicians & Surgeons, New York, New York*

**Background and objectives:** Pauci-immune necrotizing and crescentic glomerulonephritis (PNCGN) superimposed on diabetic glomerulosclerosis (DGS) is a rare occurrence. Only limited data on this dual glomerulopathy are available.

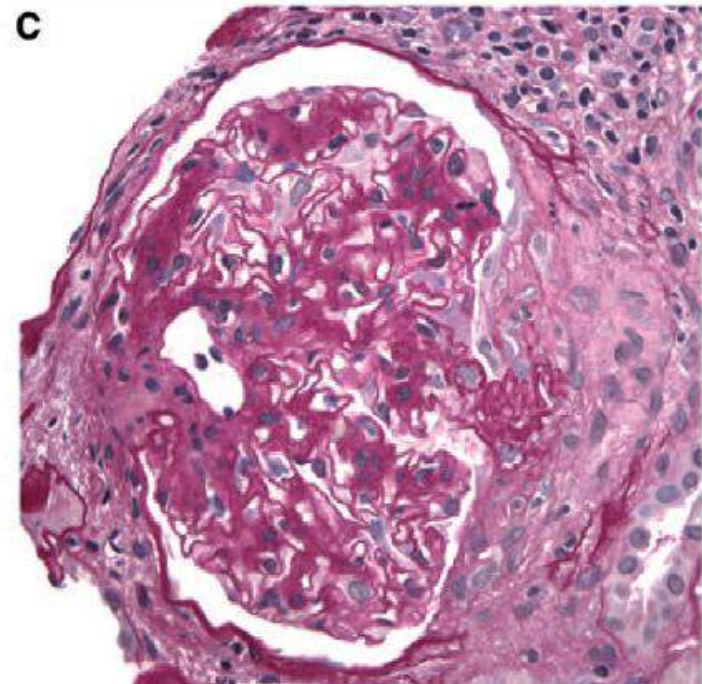
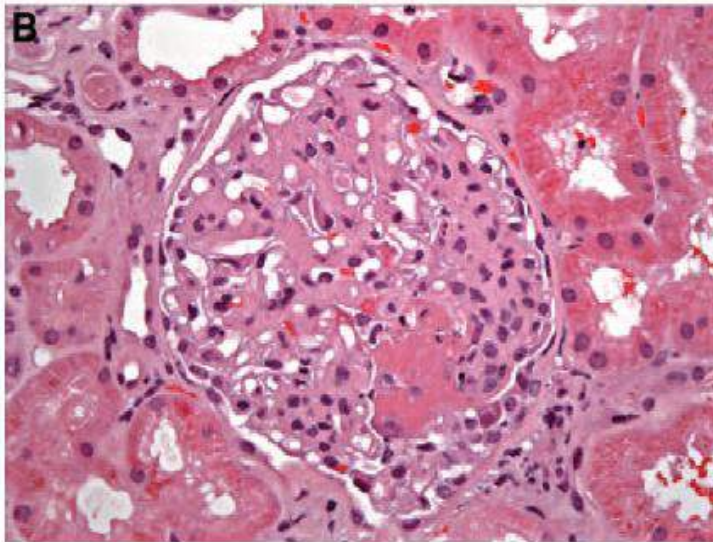
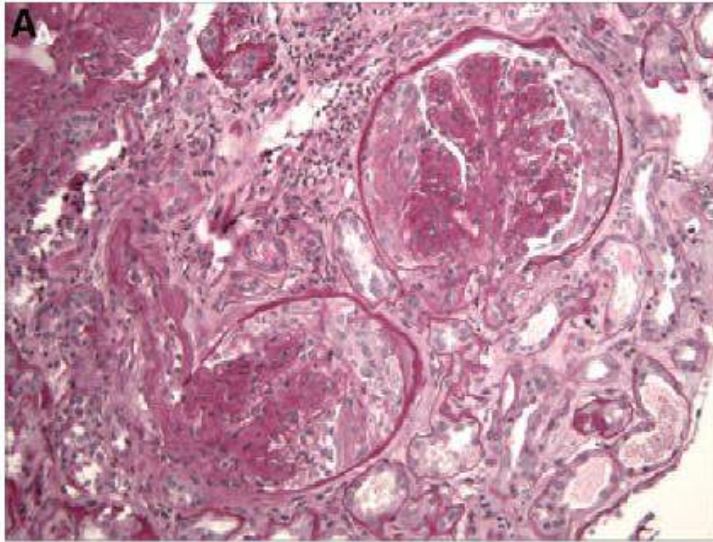
**Design, setting, participants, & measurements:** Twenty-three cases of PNCGN superimposed on DGS were identified from the archives of the Renal Pathology Laboratory of Columbia University. The clinical features, pathologic findings, and outcomes are described.

**Results:** The majority of patients were white, elderly, and had longstanding diabetes. Patients presented with acute renal failure and an active urine sediment. Antinuclear cytoplasmic autoantibody (ANCA) testing was positive by indirect immunofluorescence in 18 of 22 patients. Sixteen patients had a P-ANCA pattern, 9 of whom underwent further testing and were found to be MPO-ANCA positive by enzyme-linked immunosorbent assay. Among the two patients with C-ANCA by indirect immunofluorescence, enzyme-linked immunosorbent assay was performed in one and revealed PR3-ANCA. Eight patients had extrarenal manifestations of vasculitis, including 6 with pulmonary hemorrhage. At the time of presentation and renal biopsy, 11 patients required hemodialysis. The mean percentages of glomeruli with cellular crescents, fibrous crescents, and necrosis were 24.9, 8.4, and 12.9, respectively. Most patients were treated with cyclophosphamide and prednisone. At a mean follow-up of 14.6 mo (available in 21 patients), 8 patients had died and 8 of the remaining 13 patients had reached end-stage renal disease. Correlates of end-stage renal disease were hemodialysis at presentation and the degree of DGS.

**Conclusions:** PNCGN may occur superimposed on DGS. The prognosis for this dual glomerulopathy is dismal despite aggressive therapy.



# Diabetik Glomerüloskleroza Superpoze Olmuş Kresentik Gomerülonefrit



# Sonuç-1

- Diabetik hastalarda böbrek hastalıkları sık rastlanan, morbidite ve mortalitesi yüksek olan sekonder organ komplikasyonudur
- İdrar anormalliği ve/veya BF bozukluğu olan diabetik hastalarda sık görülen hastalık DN olmakla beraber tek neden değildir

## Sonuç-2

- İdrar anormalliđi ve/veya BF bozukluđu olan diabetiklerde non-diabetik böbrek hastaların özellikle de glomerüler hastalıkların temelde yatan hastalık olabileceđi akılda tutulmalıdır.
- Klasik klinik paradigmaya uymayan atipik başlangıç ve/veya gidiş gösteren olgularda non-diabetik böbrek hastalıkları düşünölmelidir
- Bu bağlamda uygun klinik ve lab testleri ile birlikte BBx ve histopatolojik deđerlendirme yapılmalıdır

TEŞEKKÜR EDERİM