RADYOLOJİ KIŞ OKULU 2019

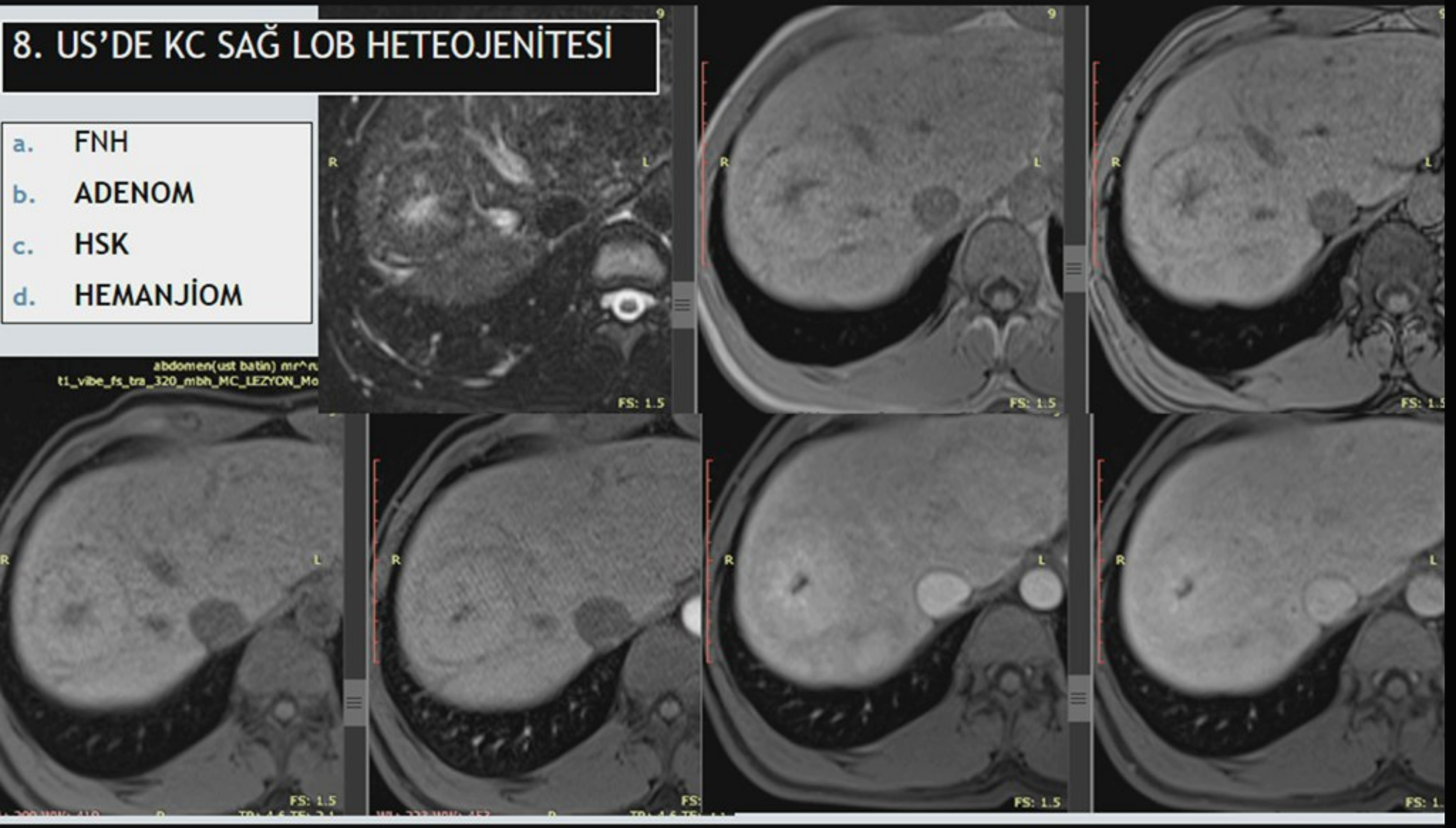
4 -10 Şubat 2019

Mirage Park Otel, Antalya

TRD 1924

8. US'DE KC SAĞ LOB HETEOJENİTESİ

a. FNH
b. ADENOM
c. HSK
d. HEMANJİOM



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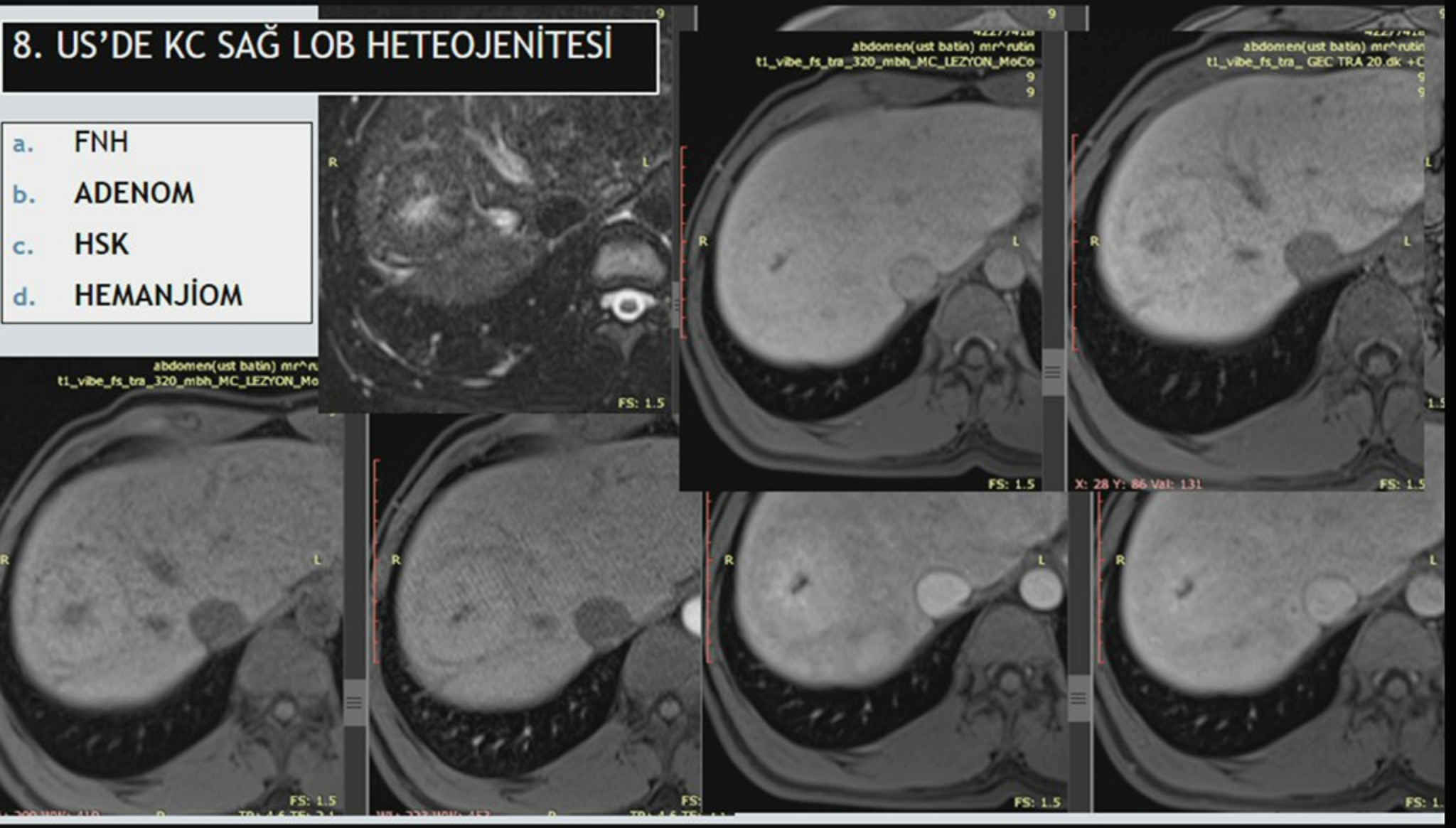
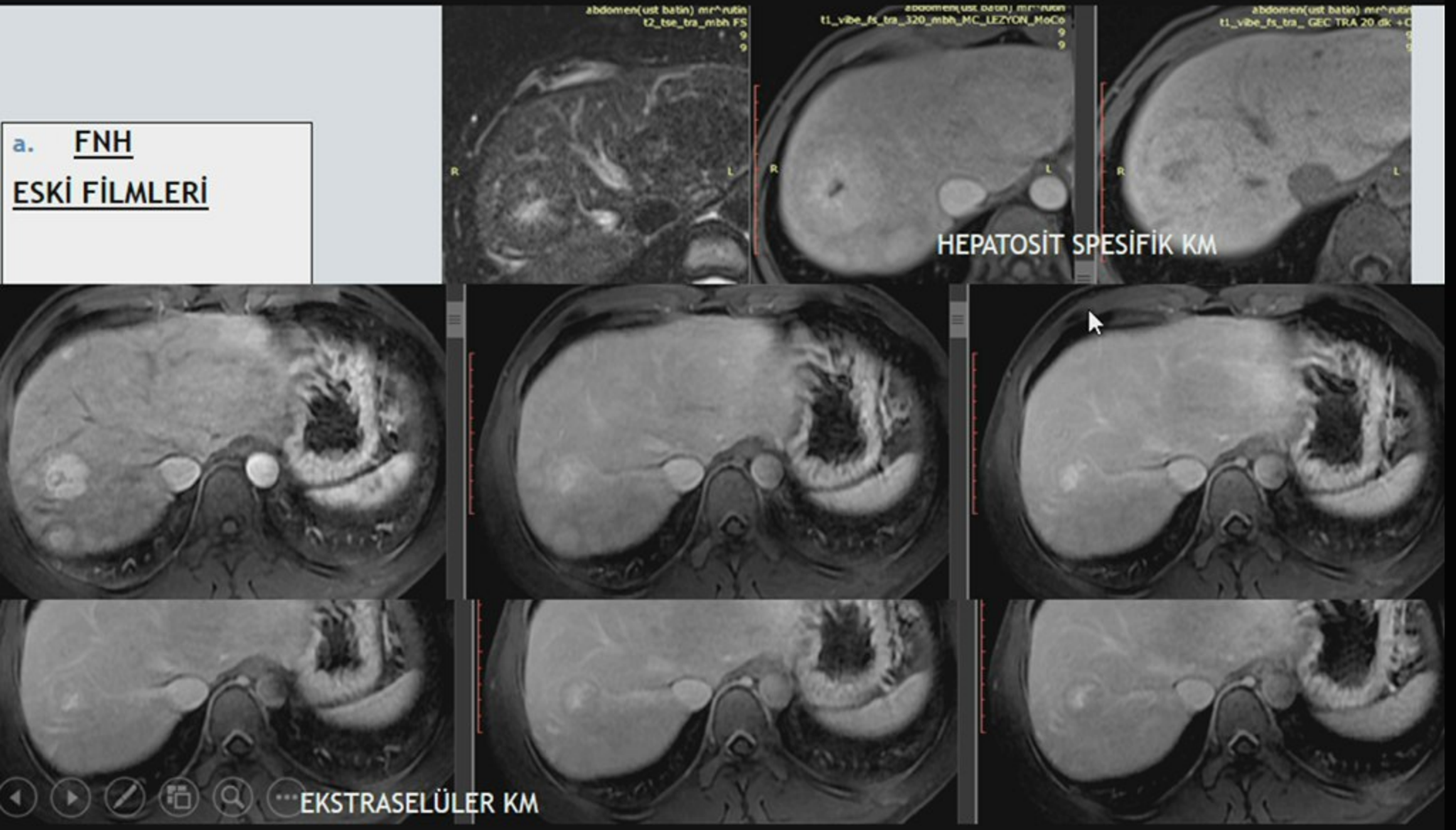


a. **FNH**

ESKİ FİLMLERİ



FNH

- İkinci en sık benign solid kc tm (%0.9)
- Yaş, cinsiyet tartışmalı
- Altta yatan neden ➛ fokal vaskuler malformasyon
- Gn.de insidental saptanır ➛ hipervaskuler diğer lezyonlardan ayrım önemli
- %70 tek lezyon, %30 ise 2-4 sayıda
- %10-20 + hemanjiom
- Ama FNH'nın malignite potansiyeli yoktur

FNH

- US: %60 hipoekoik (izo ➛ çevre vaskuler yapılar nasıl?)
- Santral skar tanıyı düşündürür ama < %20 seçilir
- RDUS: santralden perifere giden damarlar seçilir (%50 oranında araba tekerleği)
- Düşük dirençli (RI<0.5), yüksek diyastoik akım
- Tek santral besleyici arter (%77 oranında) ve drene eden ven saptanır

Semin UltrasoundCTMRI 2016; 37:511-524

FNH

- BT/MR (ESS KM):
- 5 özellik tek başlarına spesifik değildir.
- Birlikte değerlendirilirse →%95 spesifik ama bu bulguların hepsi ancak %70 olguda var olduğundan sensitivitesi azdır
  - Dansitesi/sinyali, komşu parankim ile benzer olmalı
  - Homojen
  - A fazda kuvvetli kontrastlanma göstermeli ve yıkanmamalı
  - Santral skar olmalı
  - Kapsul olmamalı

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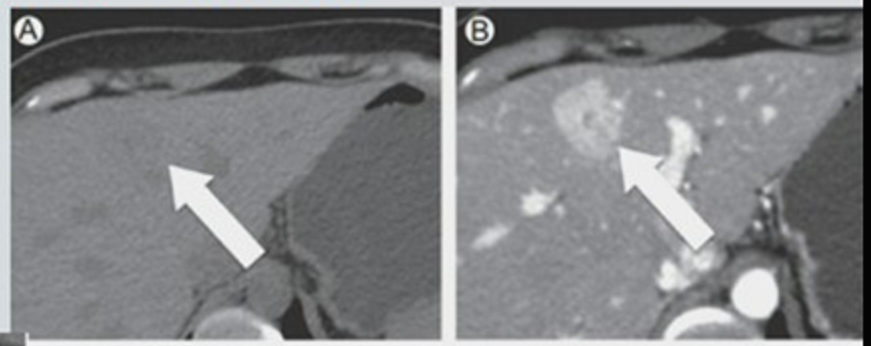
  - Santral skar olmalı
  - Kapsul olmamalı

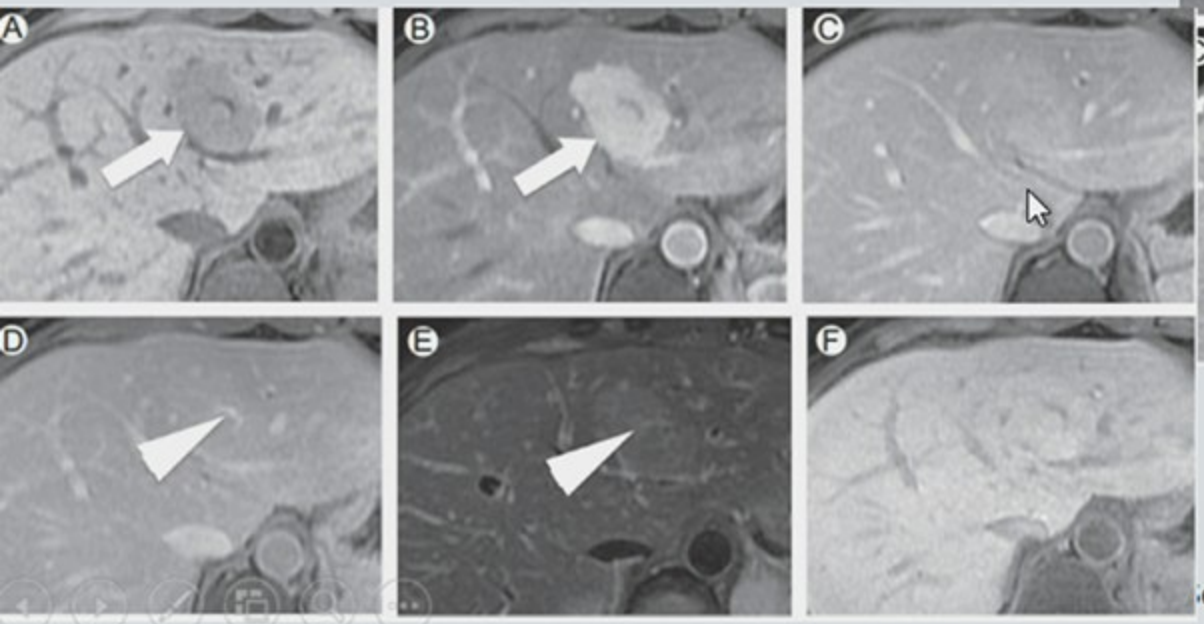
Ama unutulmaması gereken;
- Kc sirotik olmayacak
- Ekstrahepatik malignitesi olmayacak

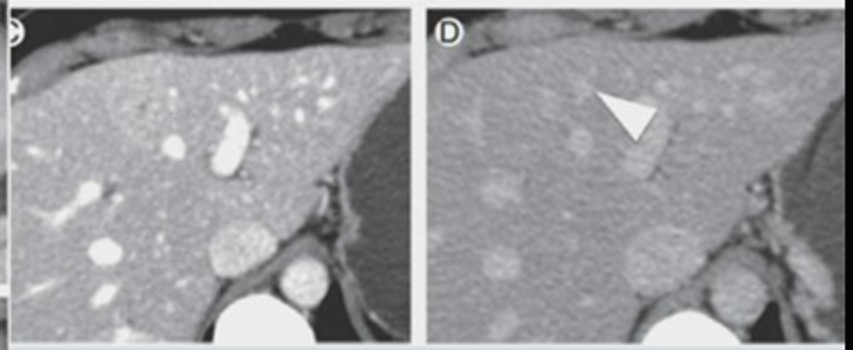
Semin Ultrasound CT MRI 2016; 37:511-524

FNH

- BT/MR (ESS KM):
  - Her zaman izodens/intens olması ?
  - A kontrastlanma → %96-100







emin Ultrasound CT MRI 2016; 37:511-524

FNH

- BT/MR (ESS KM):
  - Her zaman izodens/intens olması ?
  - A kontrastlanma → %96-100
  - Santral skar → BT ile %50, MR ile %80 gösterilir
    - T1ag hipo, T2ag hiperintens (%78-84)
    - Denge fazına doğru kontrastlanır
    - Noninvaziv tanısı için görmek şarttır (öz.le MR)
  - Lobule konturlu (kapsulu yok)
  - DWI: diğer kitlelerde ayrılamaz

Semin Ultrasound CT MRI 2016; 37:511-524

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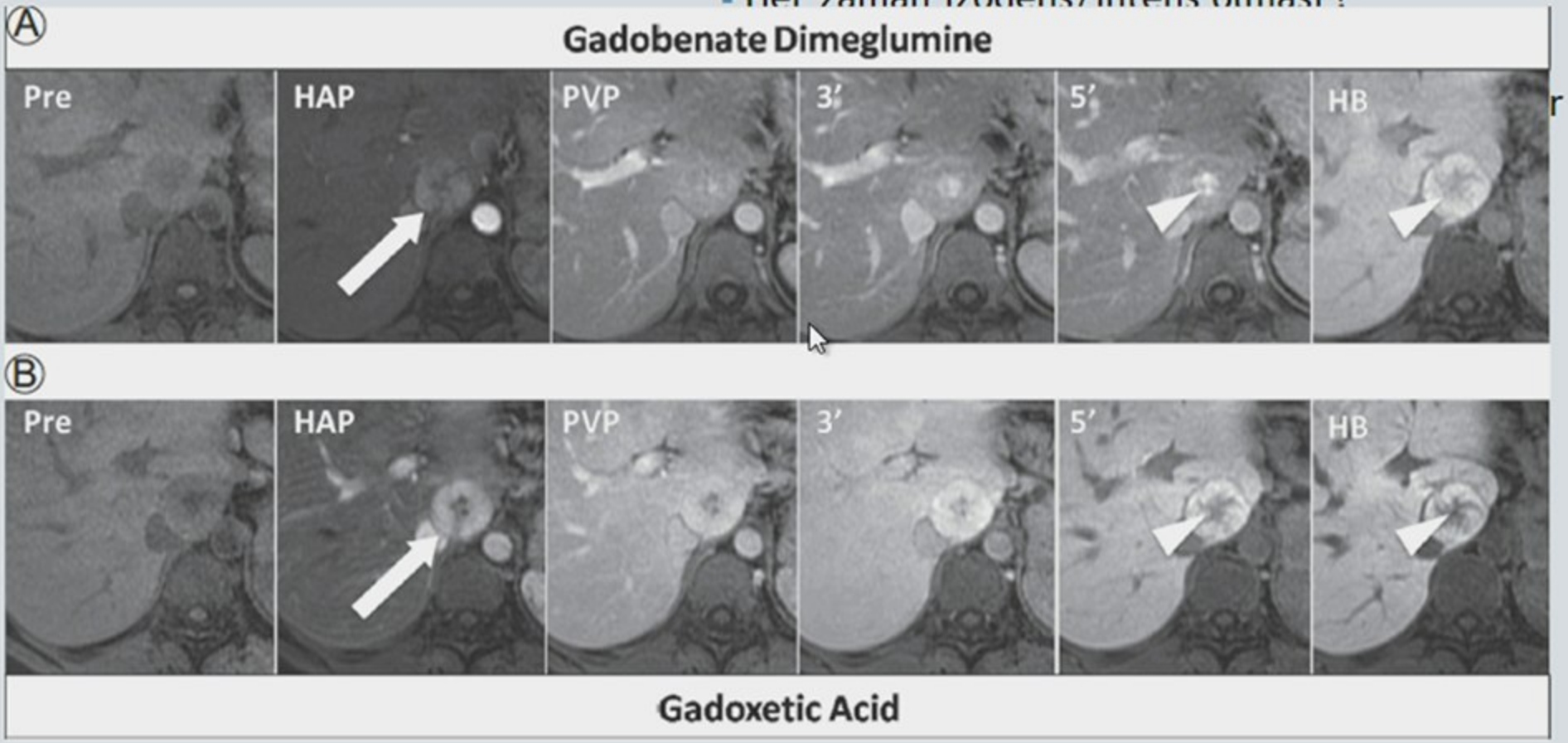
  - Her zaman izodens/intens olması ?



Semin Ultrasound CT MRI 2016; 37:511-524

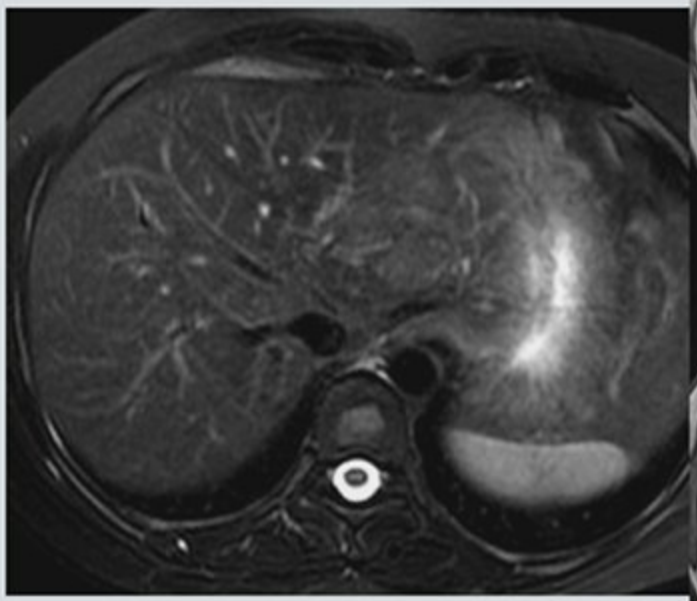
FNH

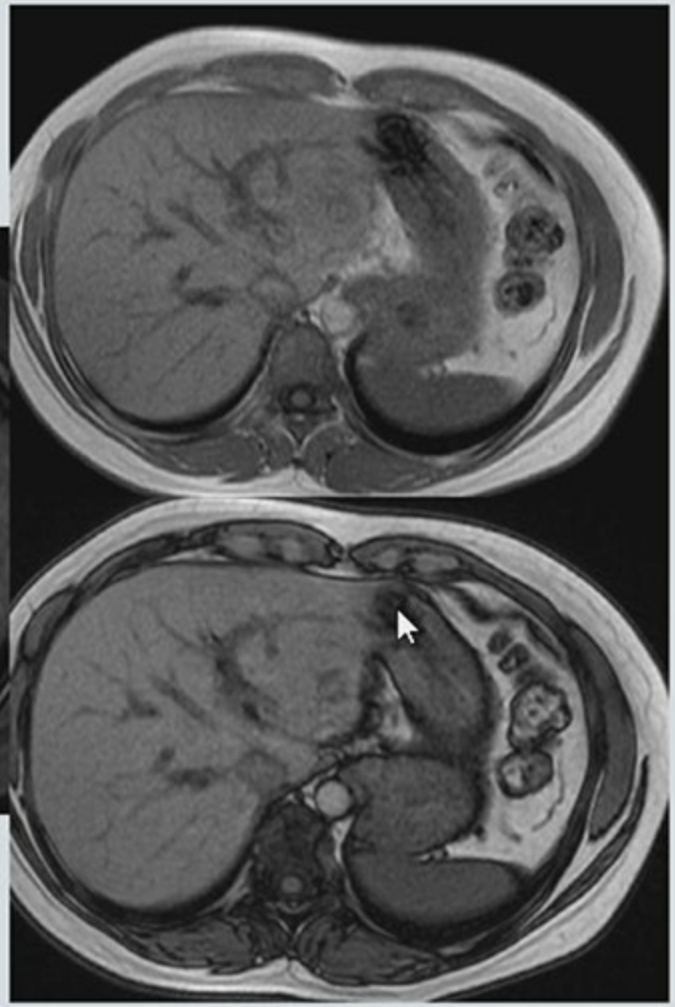
- Boyutu zamanla değişebilir (%90 küçülür)
- Kc'i ikinci sıklıktaki pedinkule lezyonudur (klasik özelliklerini genelde korur)
- Santral skar YOKSA ➛ FNH ?
  - Santral skar >3cm ➛ %65, <3cm ➛%35 oranında izlenir

Semin Ultrasound CT MRI 2016; 37:511-524

FNH

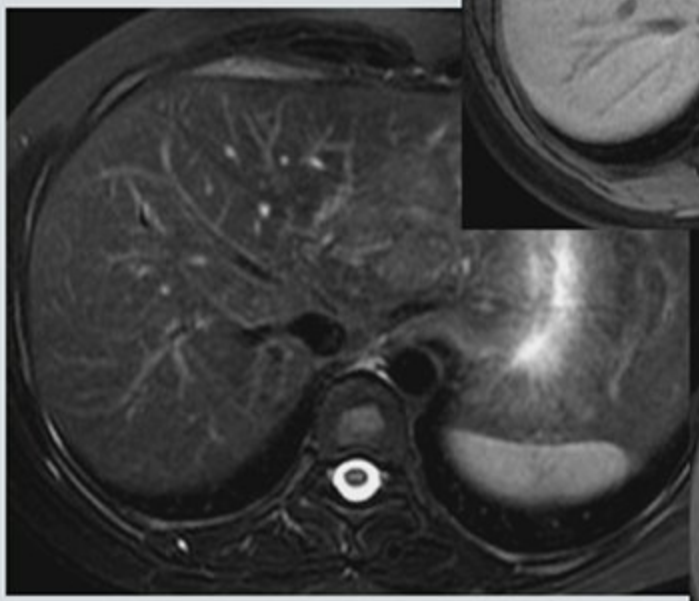
36 yaş, kadın

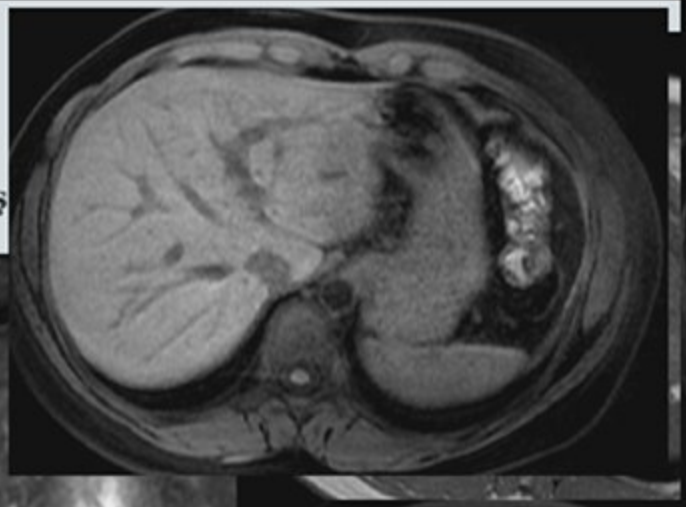


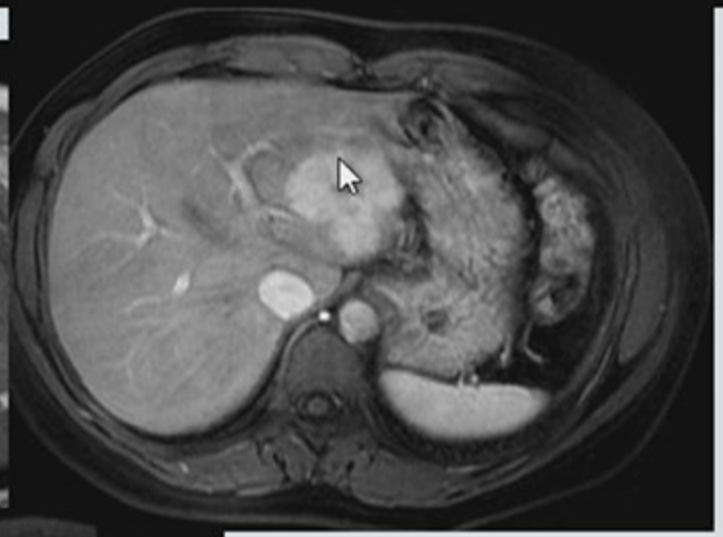


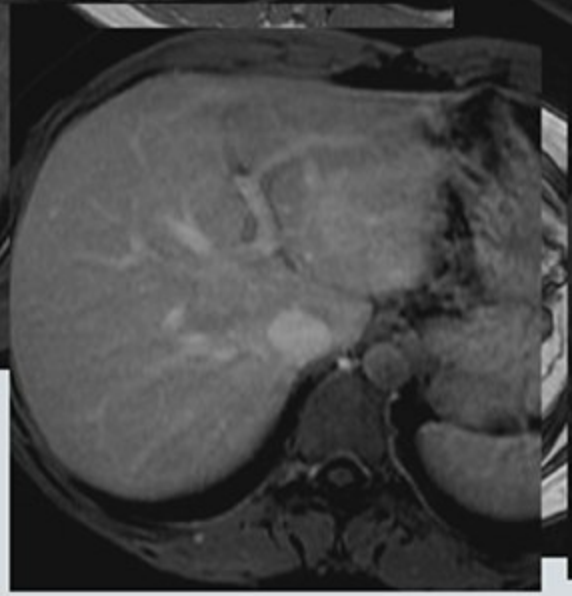
FNH

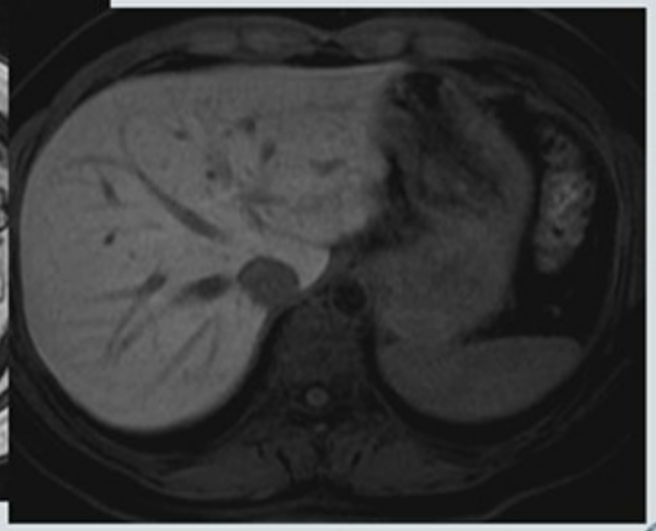
36 yaş











9. US'DE ÇOK SAYIDA KİTLE? HSK?

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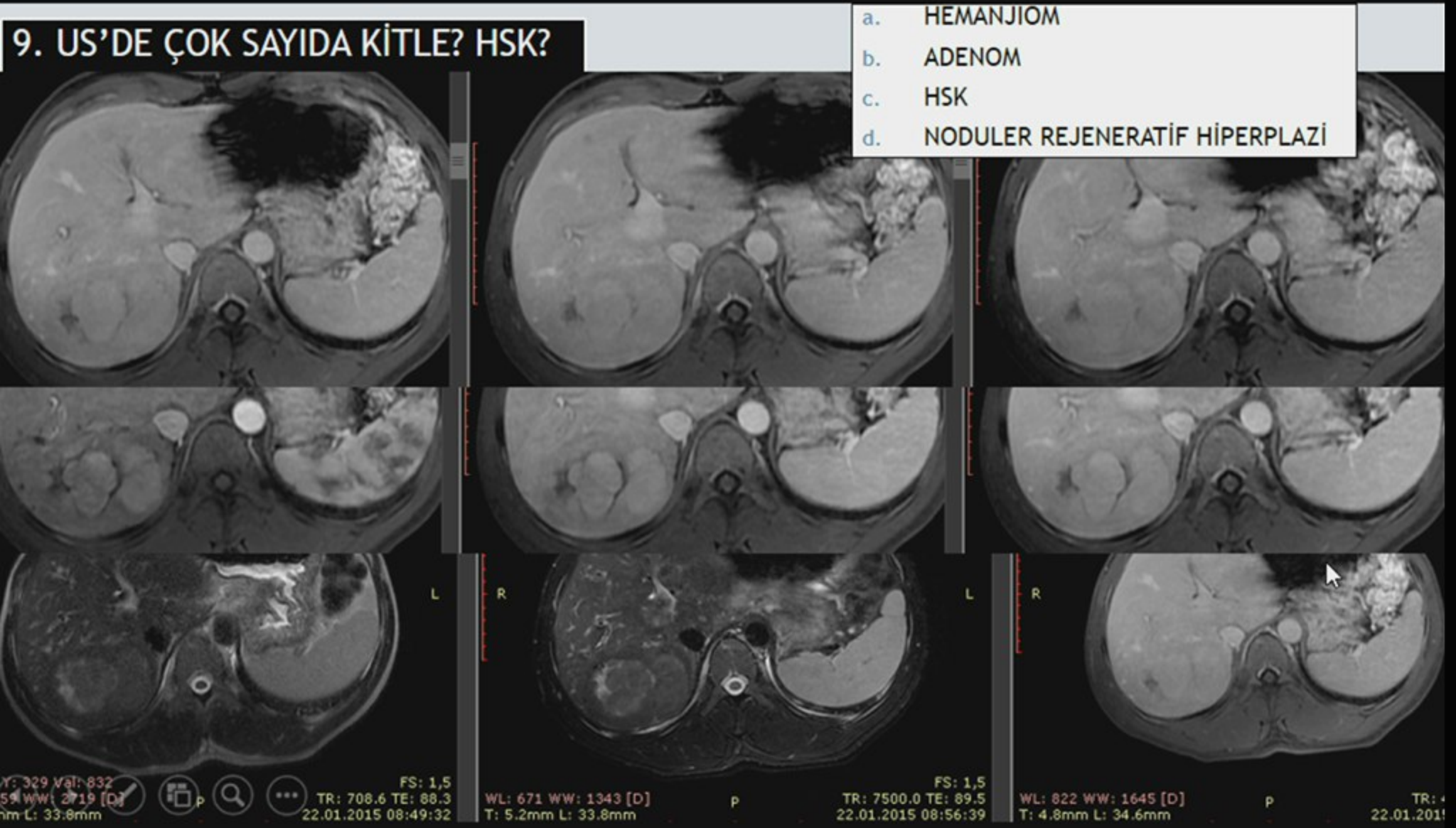
b. ADENOM
c. HSK
d. NODULER REJENERATİF HİPERPLAZİ



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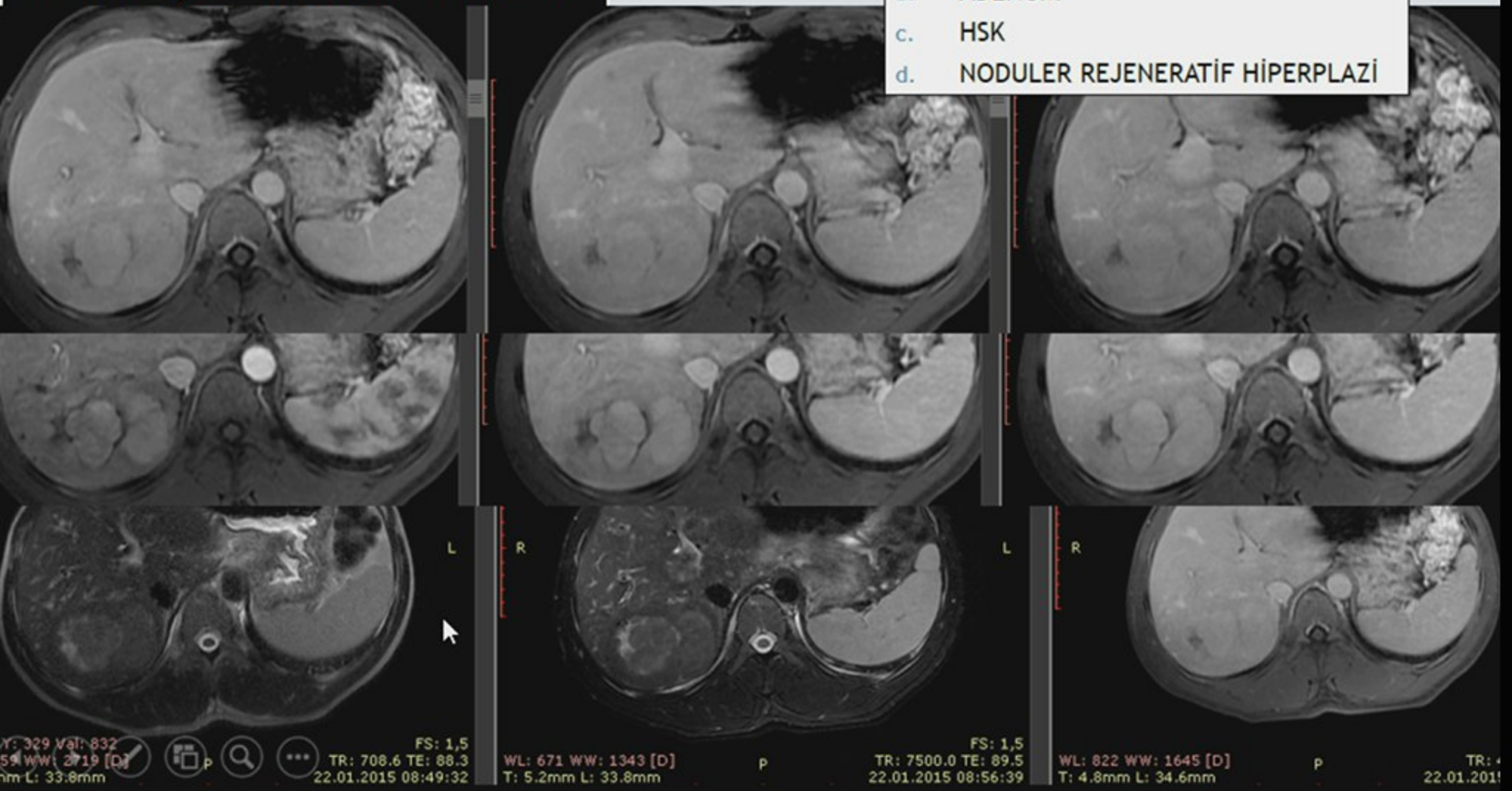

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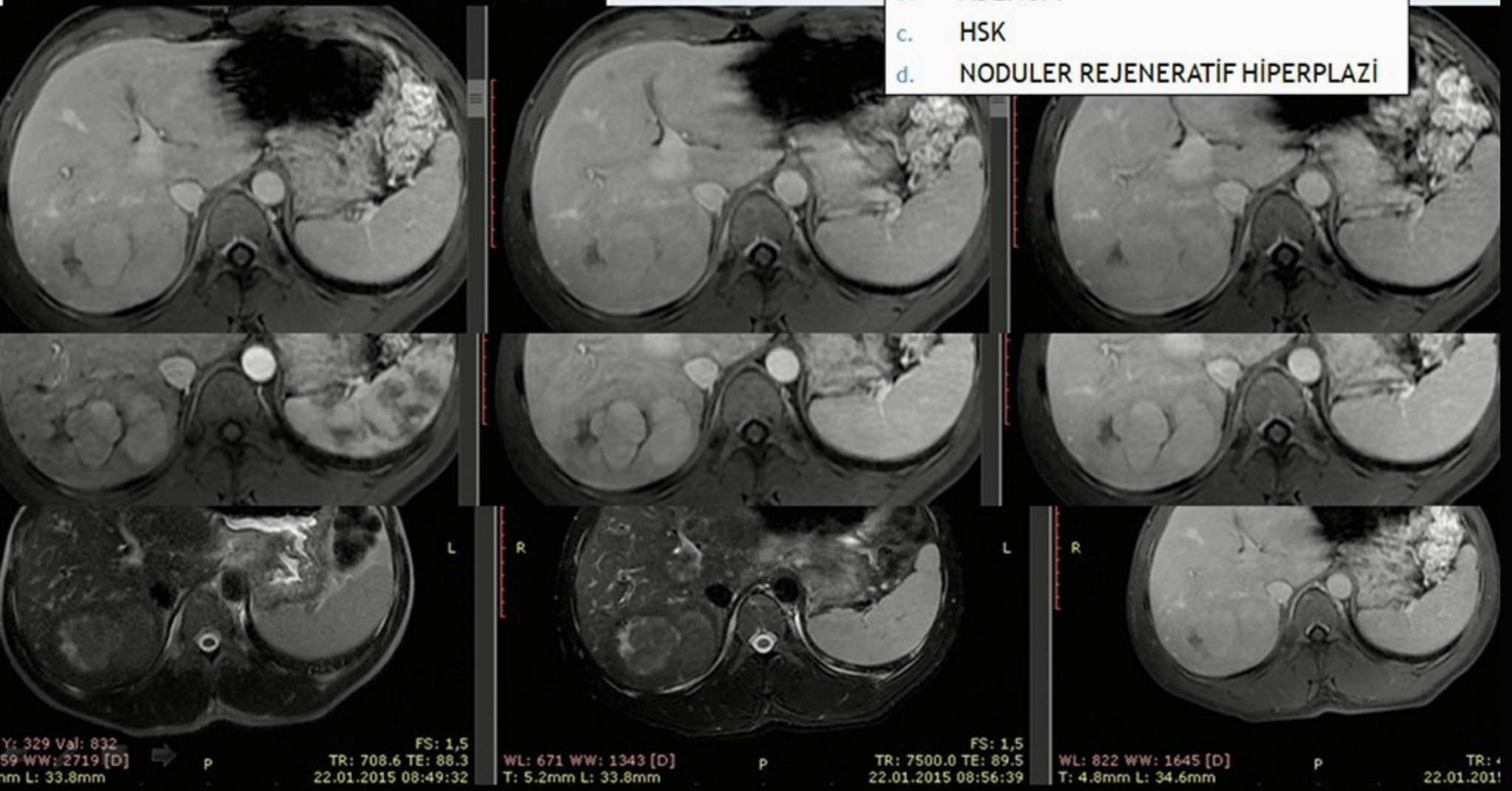

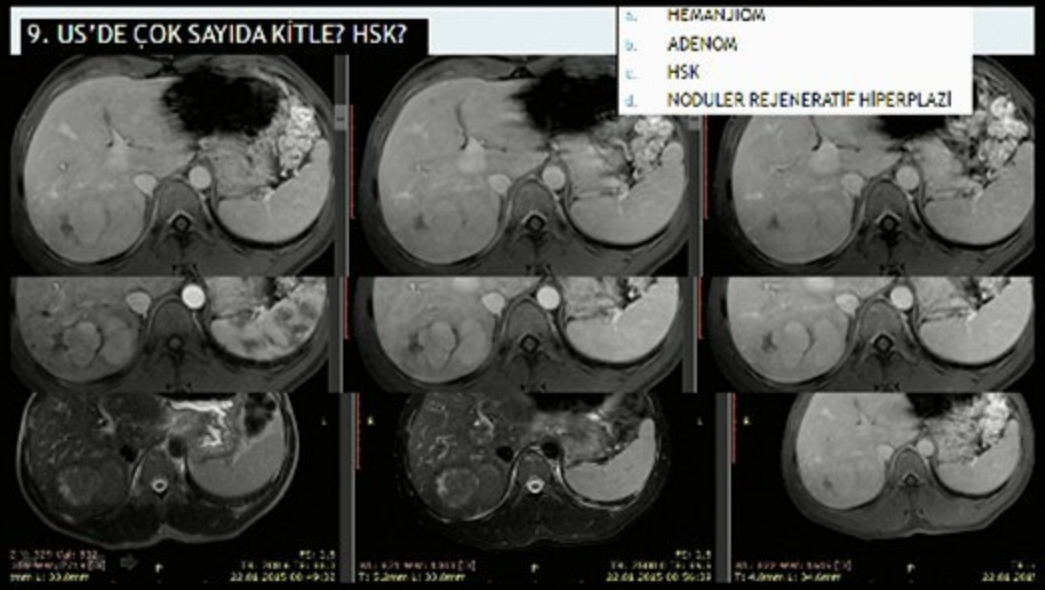
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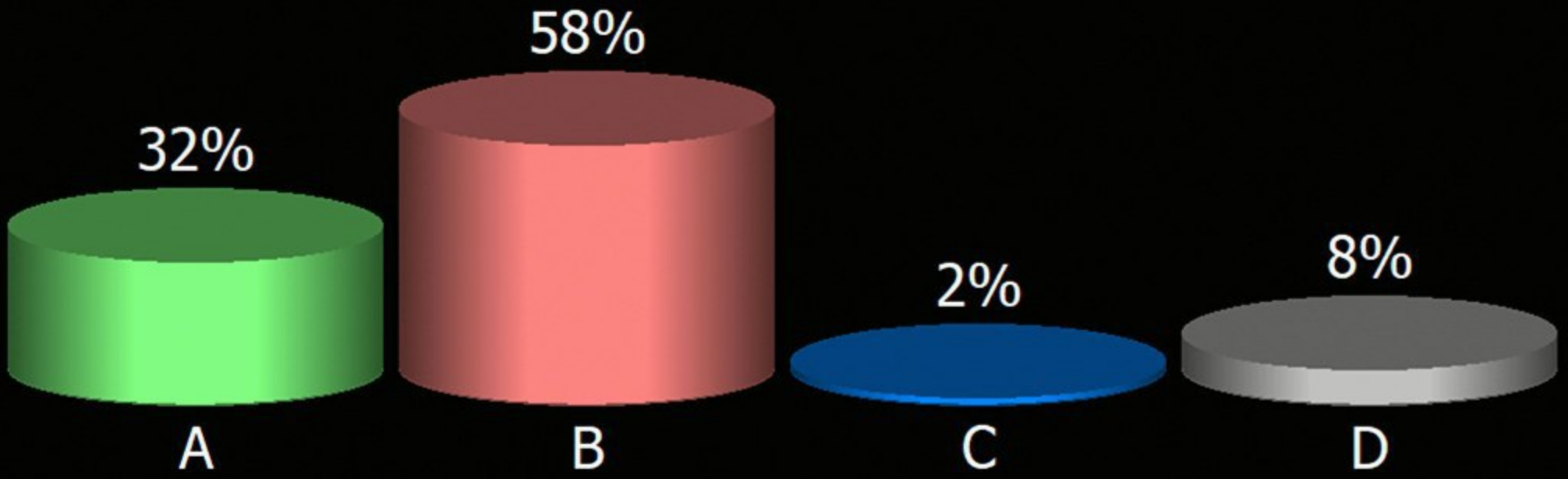
32%
58%
2%
8%
A
B
C
D

Soru: 9

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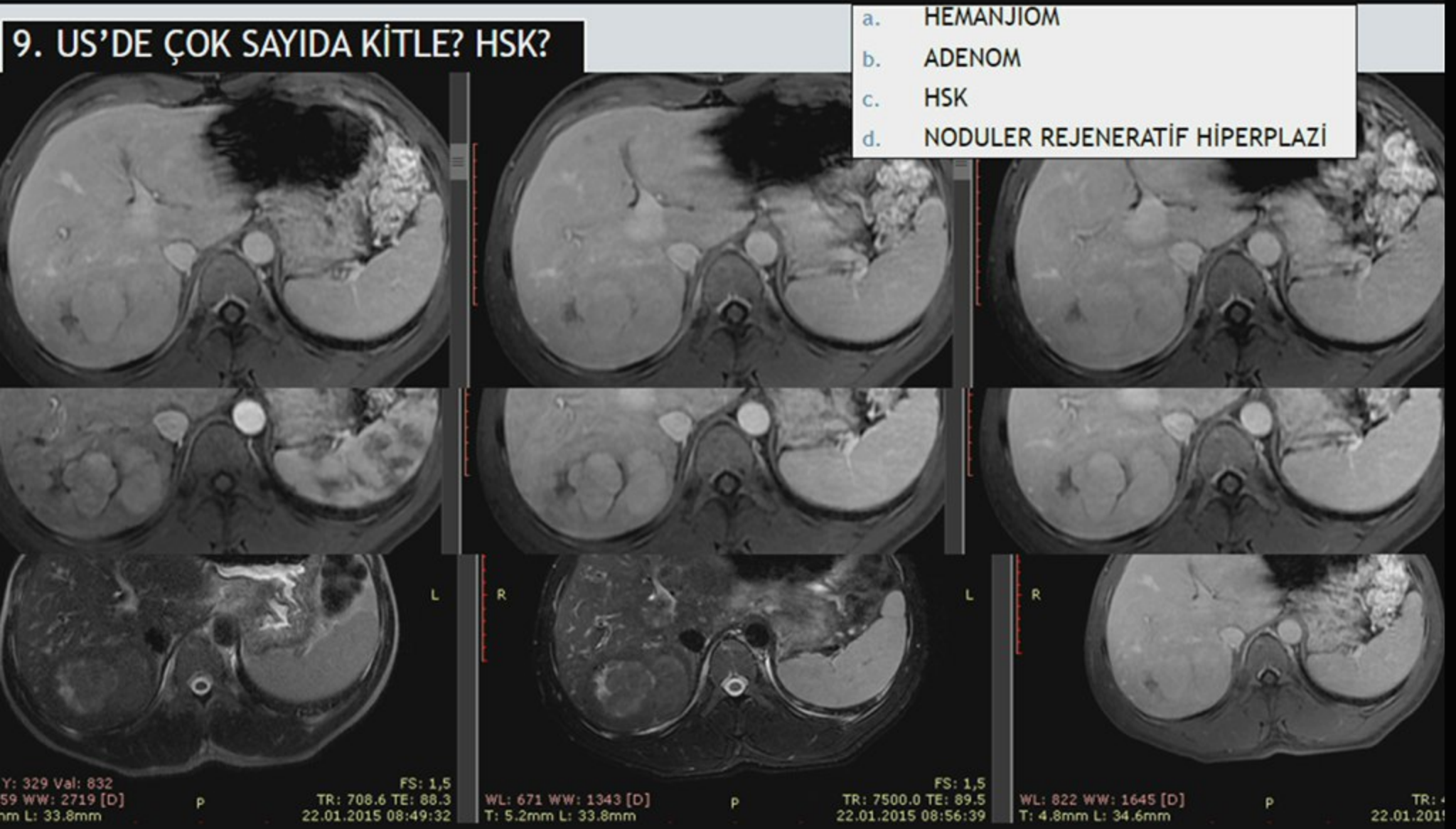

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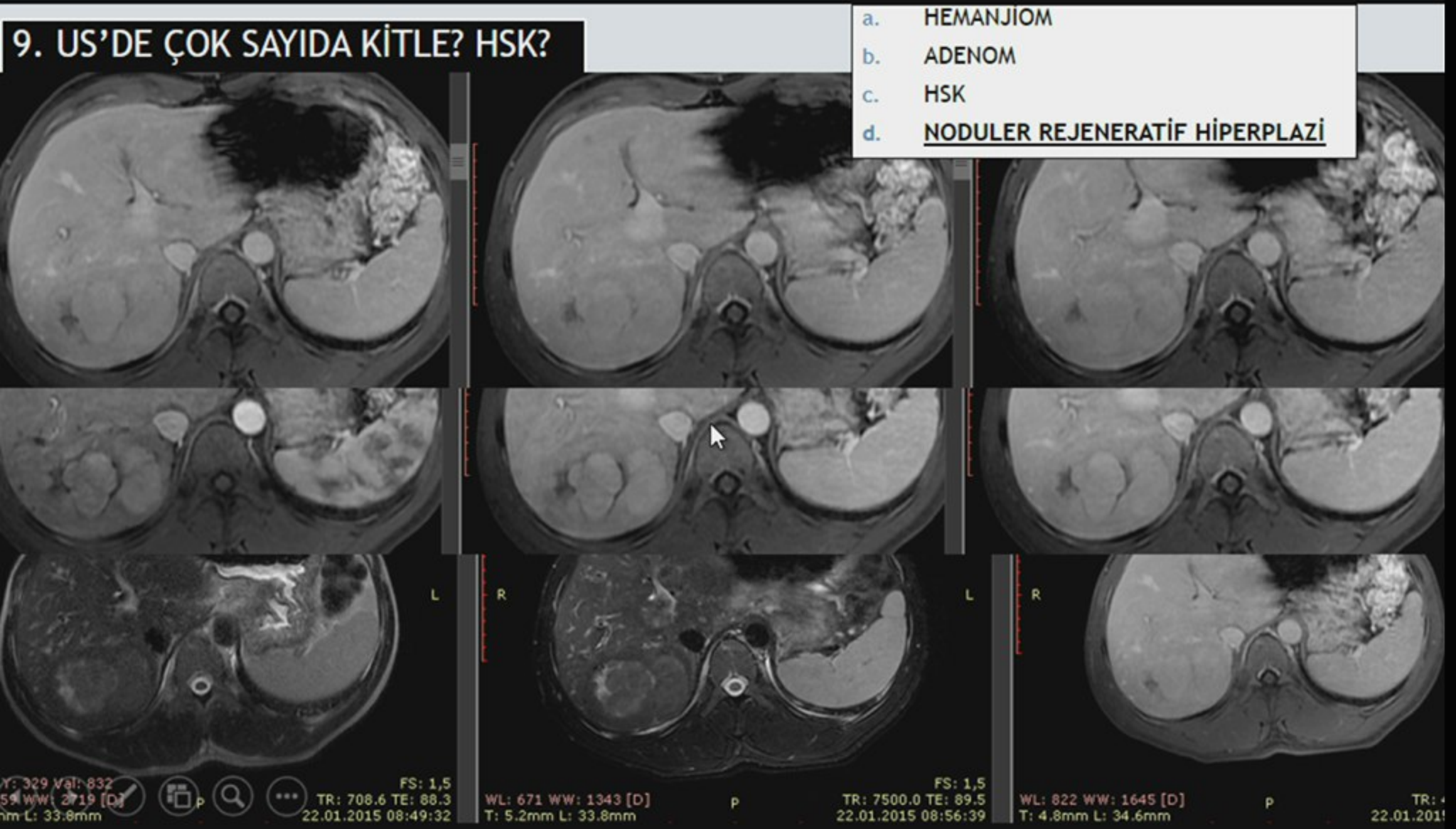

Table 1: Benign Hepatocellular Nodules Originating from Hepatocytes

Focal nodular hyperplasia (FNH)
FNH
FNH-like nodule
Serum amyloid A–positive hepatocellular neoplasm (SAA-HN)
Nodular regenerative hyperplasia (NRH)
Hepatocellular adenoma (HCA)
HNF-1α–inactivated HCA (H-HCA)
Inflammatory HCA (I-HCA)
β-catenin–activated HCA (B-HCA)
Unclassified HCA
Dysplastic nodule

Note.—HNF = hepatocyte nuclear factor.

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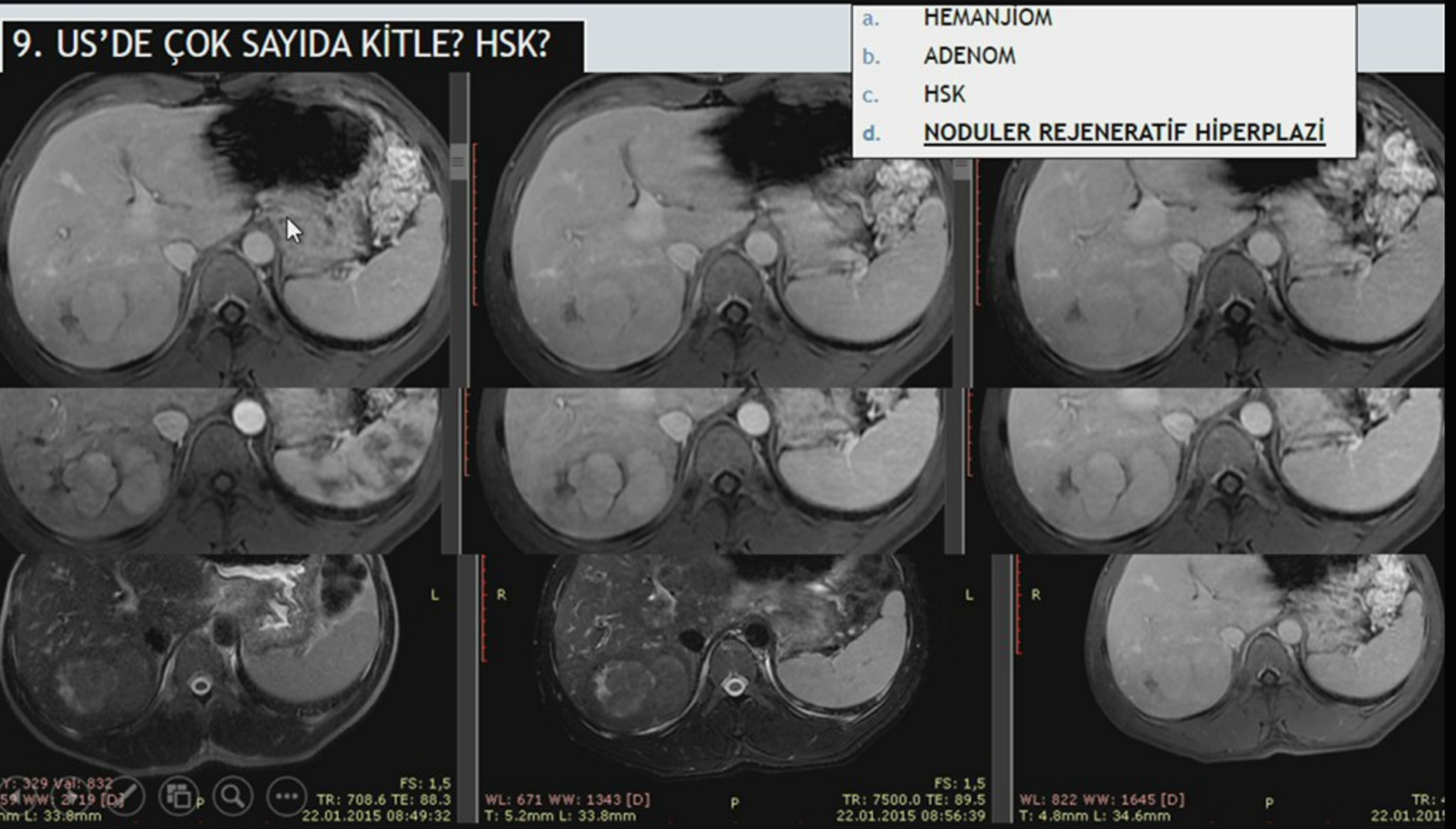

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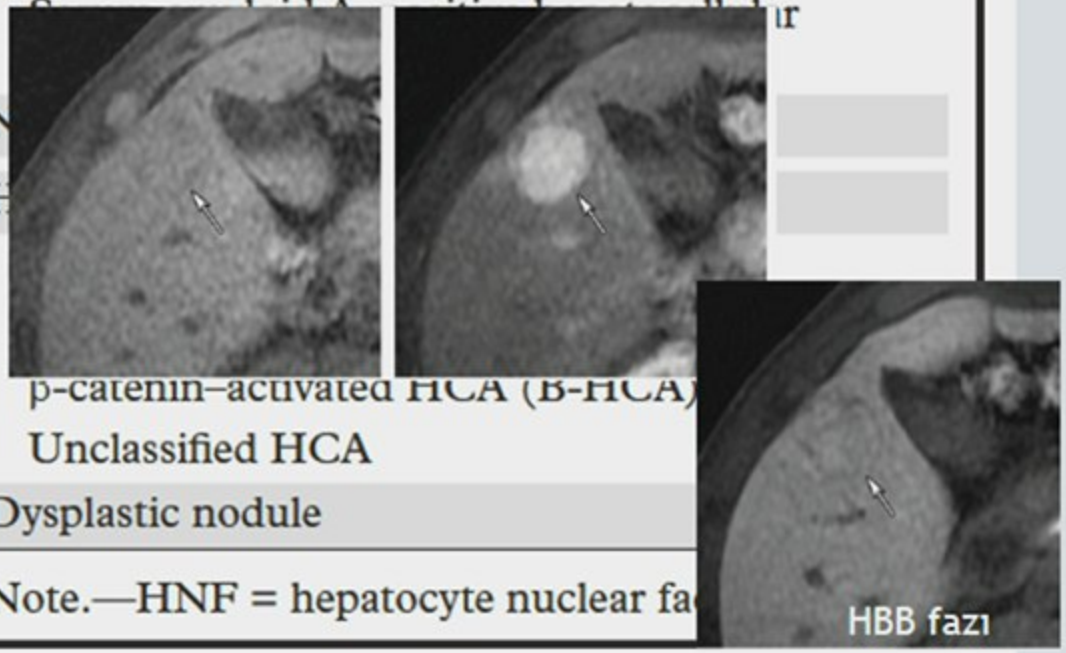

FNH-BENZERİ LEZYONLAR

- FNH- non-sirotik Kc
- FNH-benzeri- sirotik Kc
  - (öz.le alkolik siroz → HSK?)
  - BT/MR:
    - A kontrastlanır
    - geç faz yıkanma gösterir
  - T1: izo-hiper, T2: değişken
  - HBB faz: kc ile izoitens

RadioGraphics 2016; 36:2010–2027

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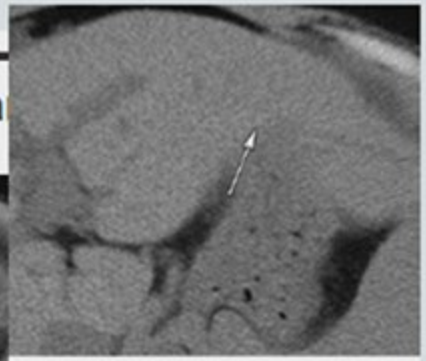
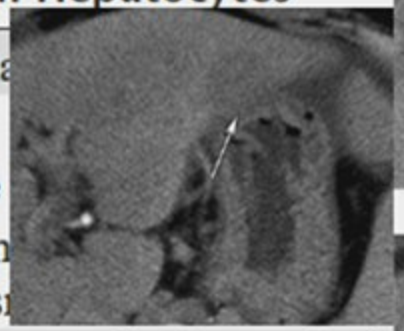
NODULER REJENERATİF HİPEPLAZİ

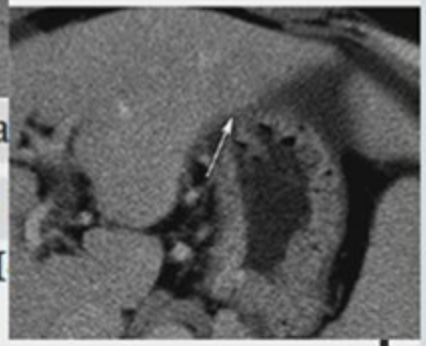
- Normal kc de görülür (otopsi →%3)
- Bud-Chiari, myeloproliferatif hast, KDH, idiopatik PHT
- Küçük (1-3mm) çok sayıda nodul (bazen büyük boyutlu)
- Fibrozis yoktur
- Nodul santralinde büyük bir portal ven seçilir

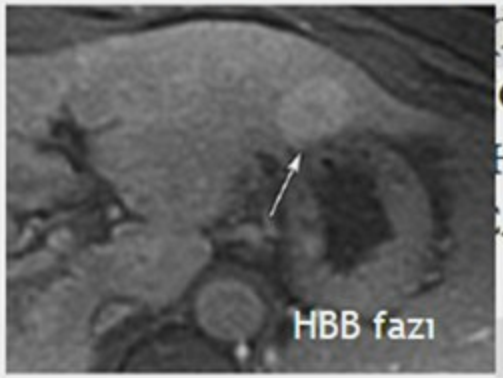
RadioGraphics 2016; 36:2010–2027

NODULER REJENERATİF HİPEPLAZİ









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Focal nodula	
FNH	
FNH-like	
Serum am	
neoplasi	
Nodular regenerative hyperplasia	
	HCA)
	CA (H
	HCA)
	A (B-HCA)

Note.—HNF = hepatocyte nuclear factor.

- Portal ven ile beslenir ➛
  - A fazda kc'e göre hipointens/dens
  - P faz: hafif kontrastlanır
  - Denge fazı: izointens/dens
  - HB faz: santrali hafif hipointens

RadioGraphics 2016; 36:2010–2027

HEPATOSELÜLER ADENOM

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Dysplastic nodule

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HEPATOSELÜLER ADENOM

- 1/1000.000
- uzun süreli OK kullanan veya kullanmış kadınlar arasında 1-3/100.000 K,
- >10 adet: hepatik adenomatozis
- Adenom benign matür hepatositler içerir, nukleer atipi ve mitoz yoktur.
- Kapsül yapısı genellikle yoktur.
- Portal ven ve safra yolu içermez.
- Klasik görünüm yağ, kan içeriği ve sinozoidal dilatasyona bağlıdır.
- Risk faktörleri:
  - OK, anebolik steroid, doğumsal hast (glikojen depo hast, DM, insülin direnci, obezite gibi metabolik sendrom

Salient characteristics of subtypes of hepatocellular adenomas (HCA).

HCA subtype (%)	Gender/Age Etiology (1)	Number	H&E criteria	Rare H&E criteria	Immunohistochemistry	Challenges in immunohistochemical interpretation	Risk for development of HCC	Genetics	Diagnosis MRI/versus biopsy
HNF1α inactivated HCA (35%)	- Females, reproductive age - Men (rare) - MODY3 (both gender)	- Solitary - Multiple -Adenomatosis	- Steatosis macro/micro - Clear cells - Ballooned cells	absence of steatosis	LFABP absent	- Weak contrast between tumor and non-tumor - LFABP not totally absent in tumor	- Low Except in absence of steatosis - MODY3	- *HNF1A* somatic (90%) - *HNF1A* constitutional (10%)	MRI good sensitivity and specificity (if steatosis present)
Inflammatory HCA (40%)	- Females, reproductive age - Men(rare) - Obesity	- Solitary - Multiple -Adenomatosis	- Sinusoidal dilatation -Inflammation -Ductular reaction	absence of sinusoidal dilatation	CRP positive	CRP+in non-tumor	Low	several mutations each leading to STAT3 pathway activation	MRI good sensitivity (if sinusoidal dilatation present)
β-catenin activated HCA (10%), exon 3 **β-catenin activated inflammatory HCA* (7%),** exon 3	- Females - Men /Females - male hormones, familial adenomatous polyposis, Glycogen storage diseases	Solitary	Cytological abnormalities may be present		-GS diffuse homogeneous -β-catenin nuclear staining		High, particularly If TERT promoter mutation	*CTNNB1* ex 3 deletions or mutations	biopsy
β-catenin activated HCA, exon 3 hotspot S45 **β-catenin activated inflammatory HCA***,exon 3 hotspot S45	- Females - Men /Females - male hormones, familial adenomatous polyposis, Glycogen storage diseases	Solitary	Cytological abnormalities may be present		-GS diffuse heterogeneous -β-cat nuclear staining rare	GS: weak or strong expression	Present	*CTNNB1* ex3 S45	Biopsy
β-catenin activated HCA, exons 7/8 **β-catenin activated inflammatory HCA***, exons 7/8	Females - Men /Females - male hormones, familial adenomatous polyposis, Glycogen storage diseases	Solitary	No Specificity		-GS focal around veins+occasional areas - no β cat nuclear staining	GS: weak or strong expression	Low	*CTNNB1* ex 7/8	Biopsy
Unclassified HCA ** (10%)	- Females - Obesity	- Solitary - Multiple	Hepatocytes may be smaller and crowded		all markers negative		Low		Biopsy

*CRP+

**Recently "shHCA" due to dysregulation of prostaglandin pathway have been identified. Their identification by immunhistochemistry is not yet validated.[12]

LFABP: liver fatty acid binding protein; CRP: C Reactive Protein; HCC hepatocellular carcinoma; MRI, magnetic resonance imaging; MODY: maturity onset diabetes of the Young.

(1) All HCA subtypes occur mainly in women of reproductive age taking oral contraceptives but can be observed in men, in children, and in older individuals (after menopause in women). Some subtypes are more frequent in patients with high body mass index (Inflammatory HCA, unclassified HCA), MODY 3 (HNF1α inactivated HCA) and in patients exposed to male hormones (β-catenin exon 3 activated HCA). In patients with glycogen storage diseases, all subtypes can be seen except HNF1α inactivated HCA.

HEPATOSELÜLER ADENOM

Imaging Features	H-HCA	I-HCA	B-HCA	Unclassified HCA
Typical features	Diffuse signal dropout on out-of-phase T1WI	Hyperintense rim at periphery of lesion on T2WI (atoll sign)	Intralesional scar	...
Hemodynamics	Mild to moderate arterial hypervascularity	Strong arterial hypervascularity	Mild to moderate arterial hypervascularity	Variable
HB phase	Hypointense	Hypointense (some hyperintense)	Iso- or hyperintense	Hypointense

Note.—T1WI = T1-weighted images, T2WI = T2-weighted images.

- Bunların 3'ünde malignite potansiyali yüksek
  - ✓HNF 1 Alfa gen mut. *(HA-H): %30, yağ +, ailevi, soliter/adenomatosis send. Malignite riski düşük
  - ✓Enflamatuar Adenom *(HA-I): %35, obez, soliter/adenomatosis send, T2 hiper, atoll işareti, yağ ±, HBF hipo
  - ✓Beta catenin mut.(HA-B): %10, E>, gn.de hormon alımı ile ilgili, soliter, hemoraji+, skar+, HBF izo,
  - ✓Unklasifiye HCA: %10*, atipik bulgular

HEPATOSELÜLER ADENOM

- **M dejenerasyon için**:
  - E+ metabolik sendrom= 10x K göre kanserleşme riski
  - Androjen kullanımı
  - >5cm
  - Beta katenin mutasyonu gösteren histopatolojik tipi

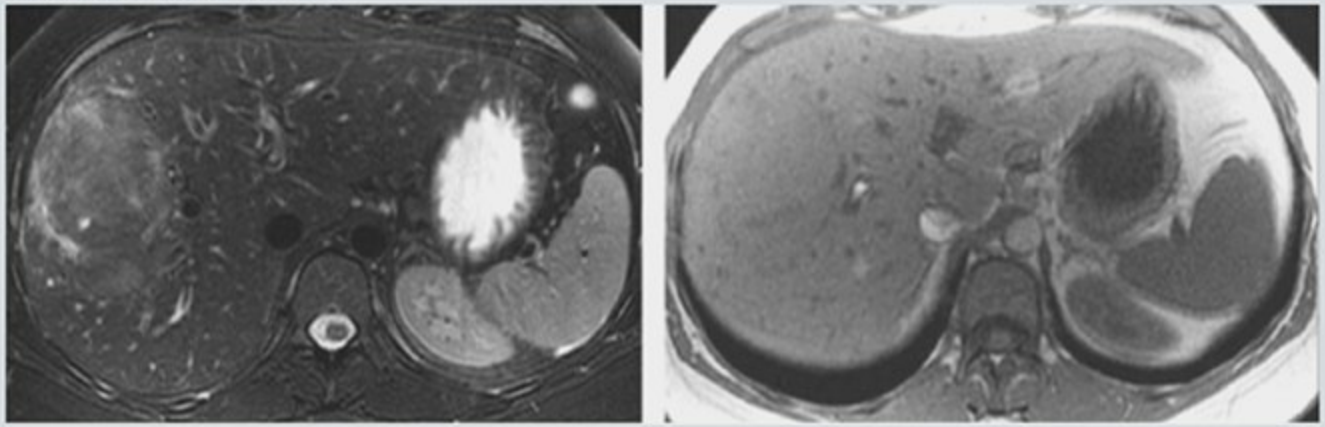
  - **HA ile FNH ayrılması gerekir**
    - HBB faz:
      - HA → hipointens (kc'e göre uptake azdır)
      - FNH → izo veya hiperintens

HEPATOSELÜLER ADENOM

- **M dejenerasyon için**:
  - E+ metabolik sendrom= 10x K göre kanserleşme riski
  - Androjen kullanımı
  - >5cm
  - Beta katenin mutasyonu gösteren histopatolojik tipi

  - **HA ile FNH ayrılması gerekir**
    - HBB faz:
      - HA ➛hipointens (kc'e göre uptake azdır)
      - FNH ➛ izo veya hiperintens

HEPATOSELÜLER ADENOM



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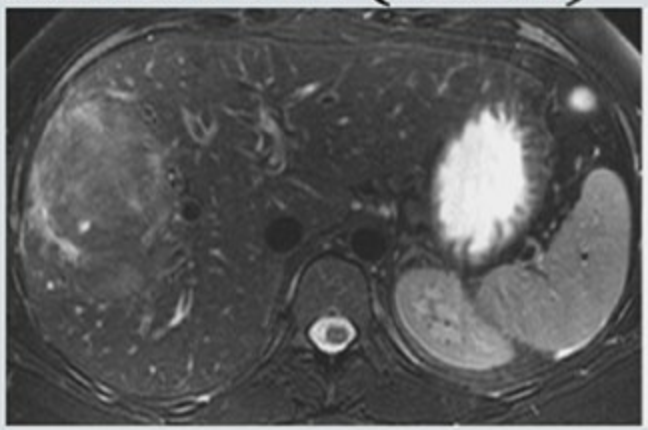
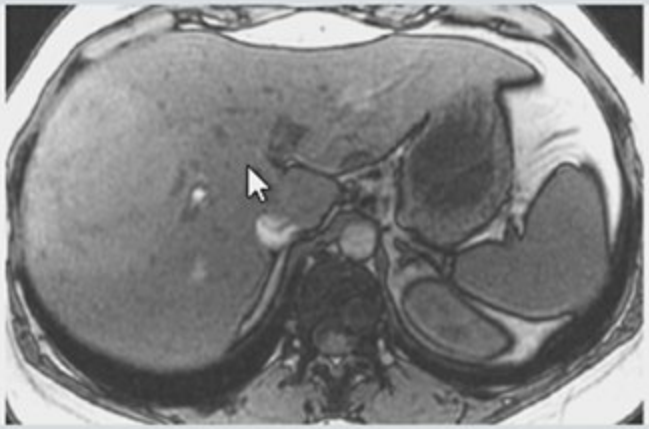
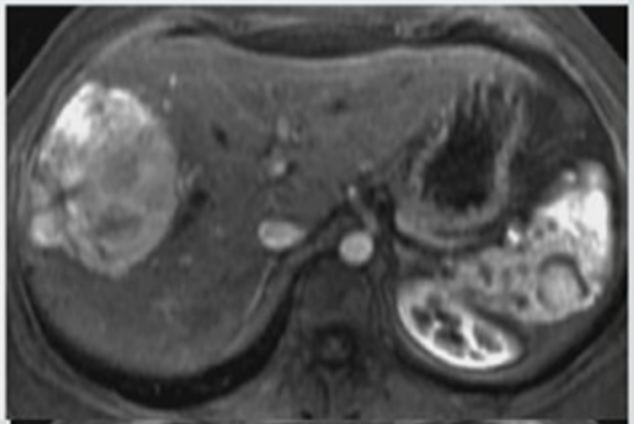
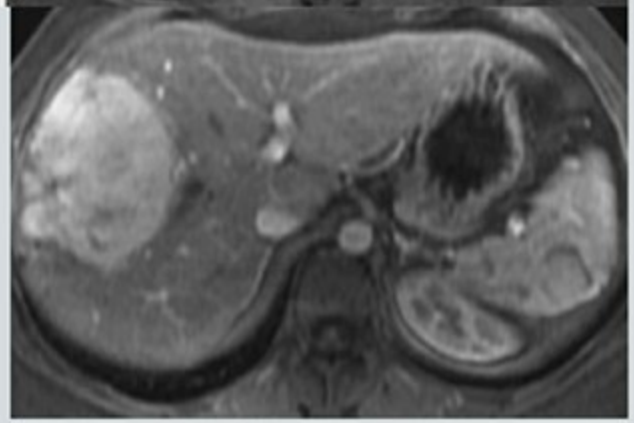
HEPATOSELÜLER ADENOM (HA-I)









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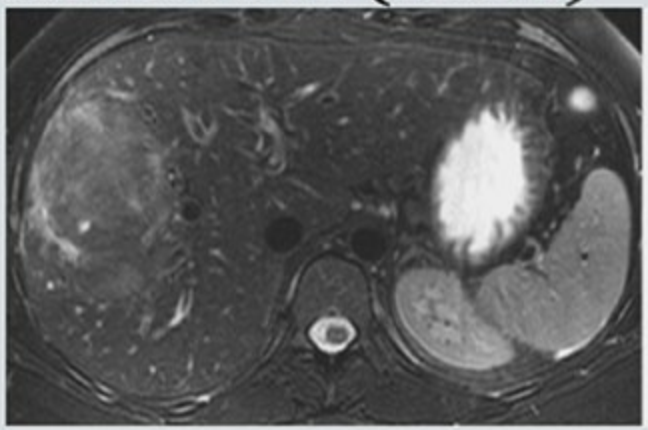

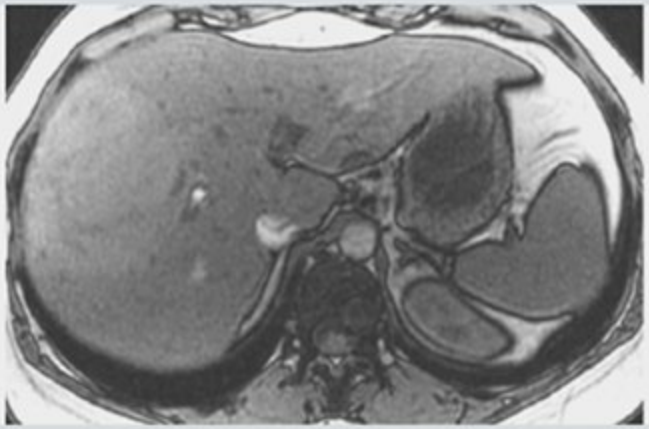

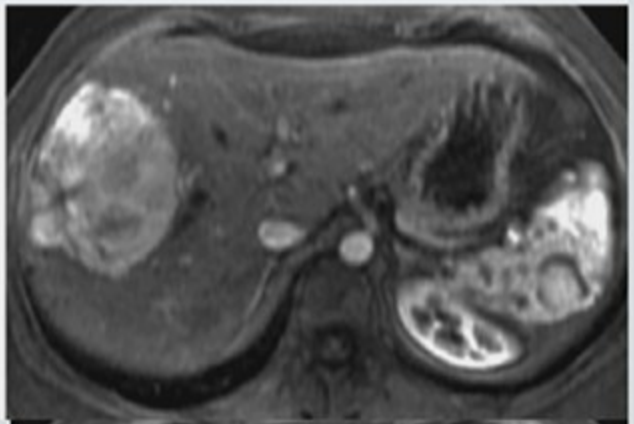

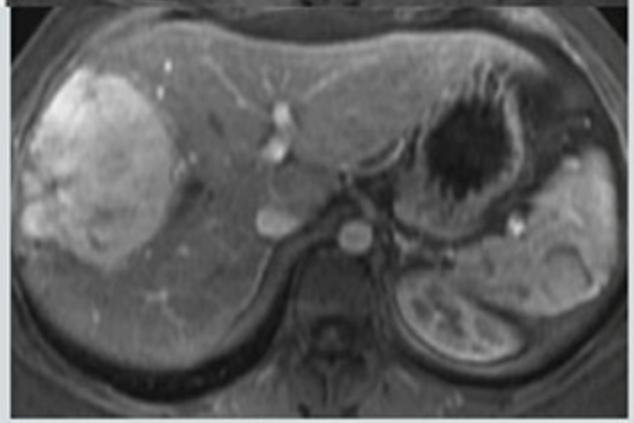

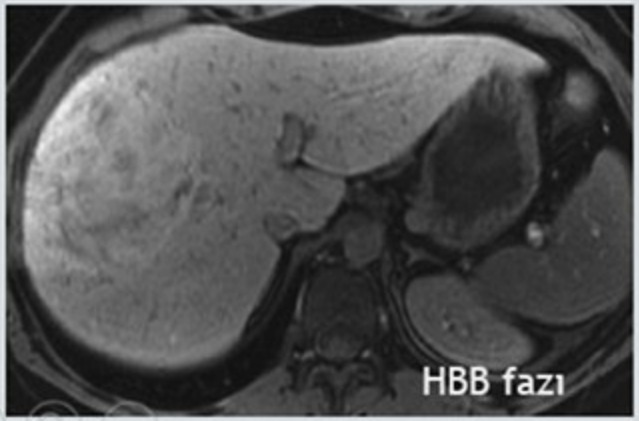

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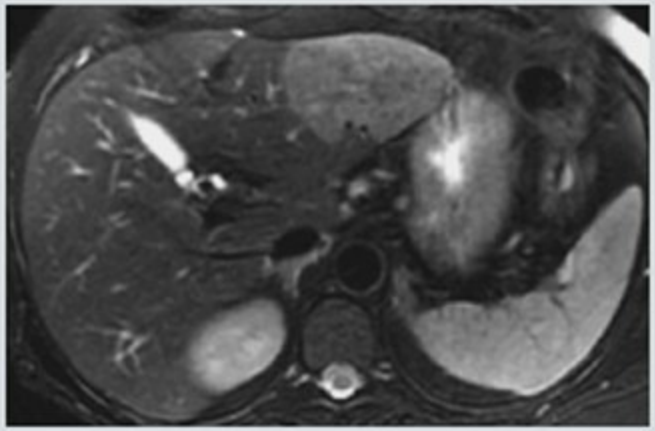
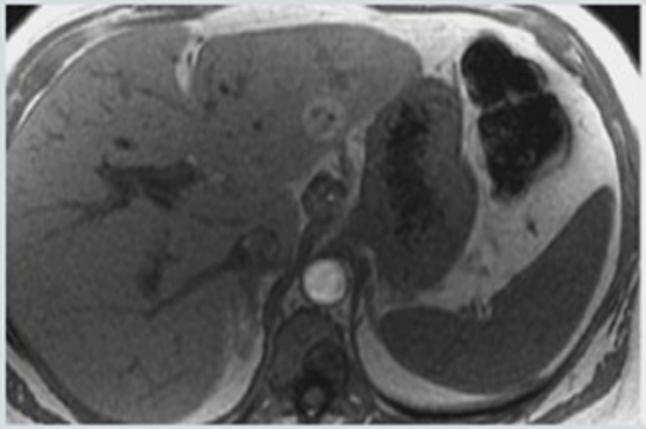
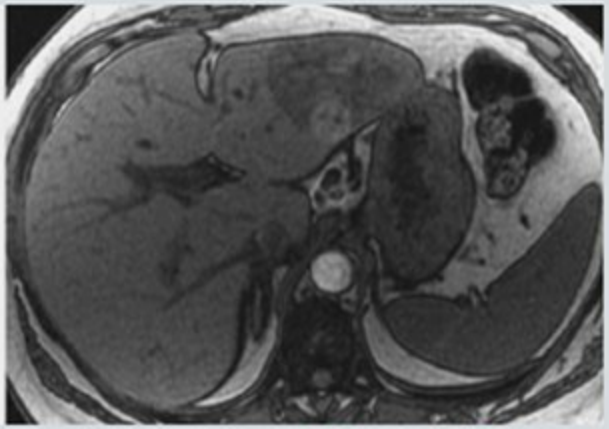
HEPATOSELÜLER ADENOM (HA-I)







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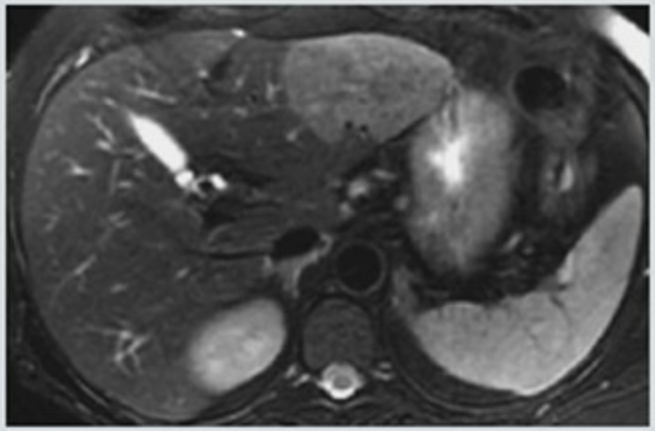
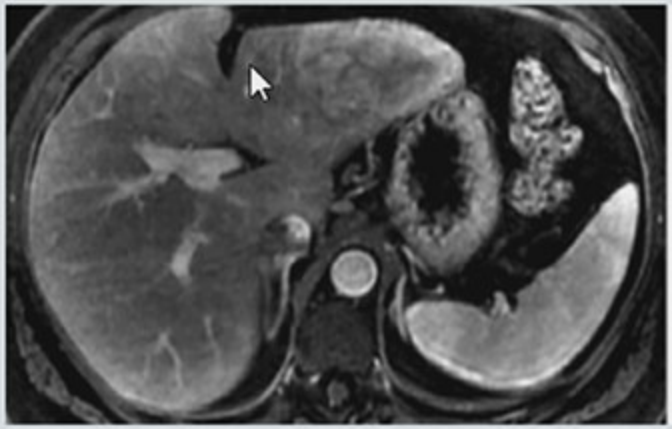
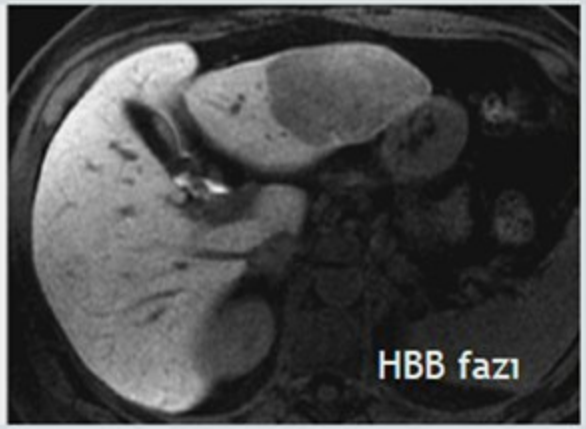
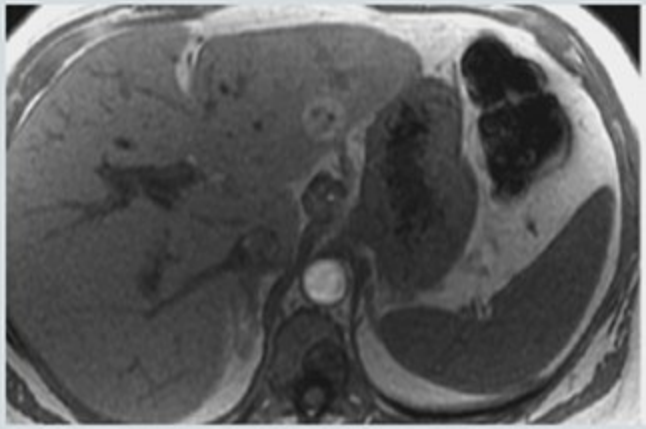
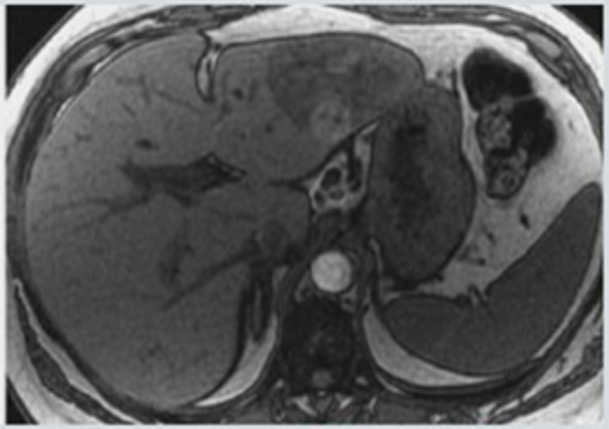
HEPATOSELÜLER ADENOM (HA-I)











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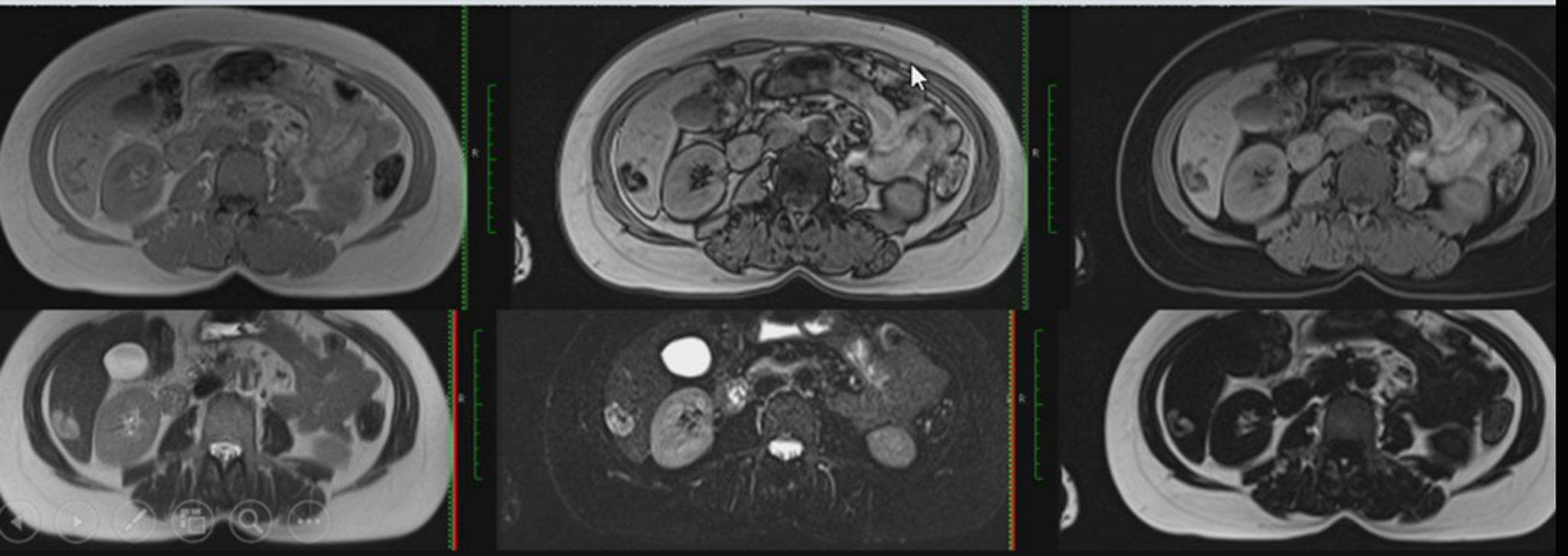
10. TANINIZ EDİR?

a. HEMANJİOM
b. HEPATOSELÜLER ADENOM
c. HSK
d. NODULER REJENERATİF HİPERPLAZİ



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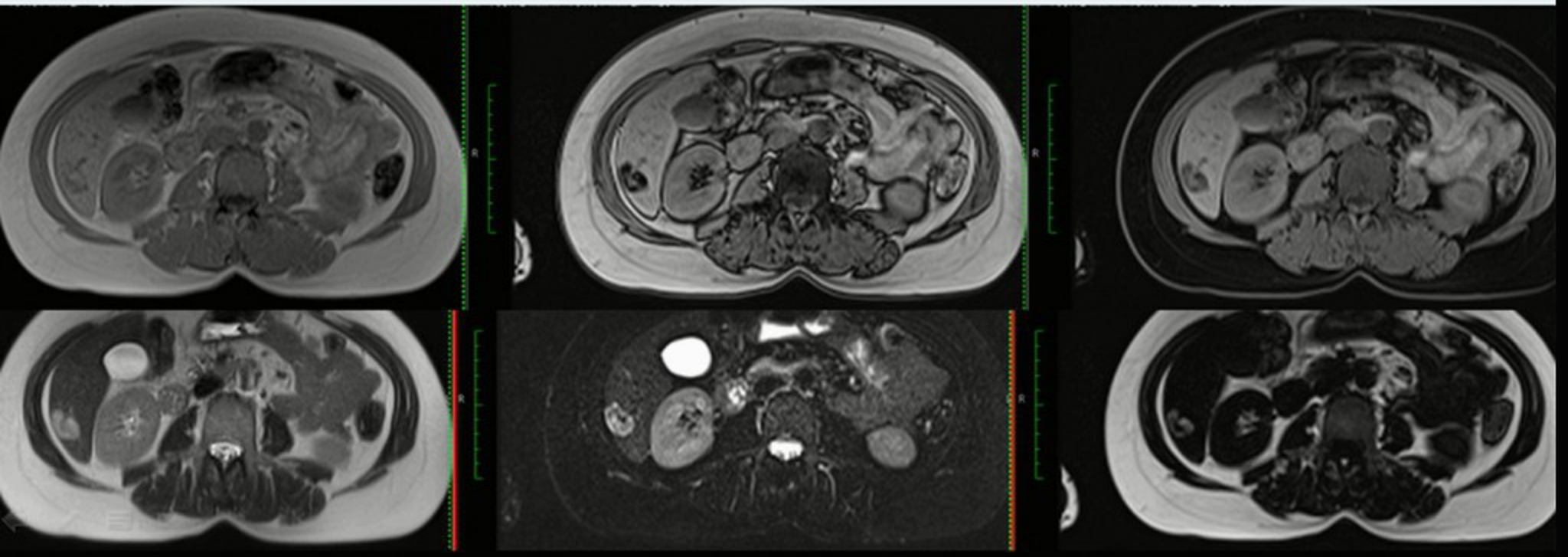

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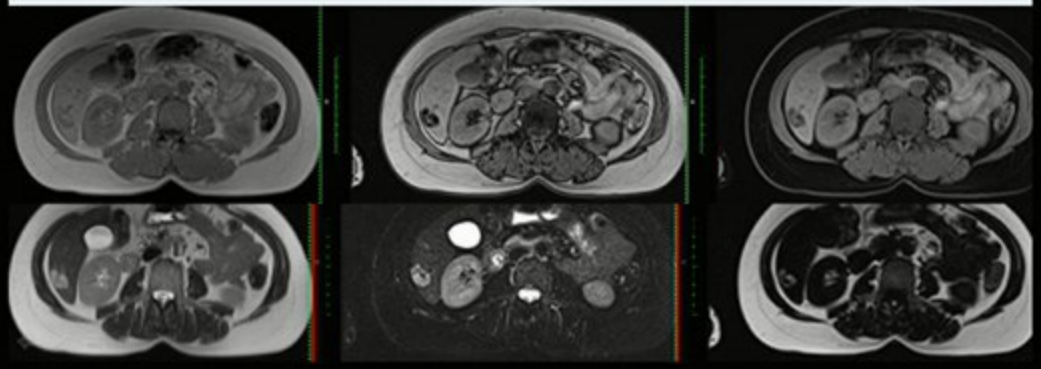

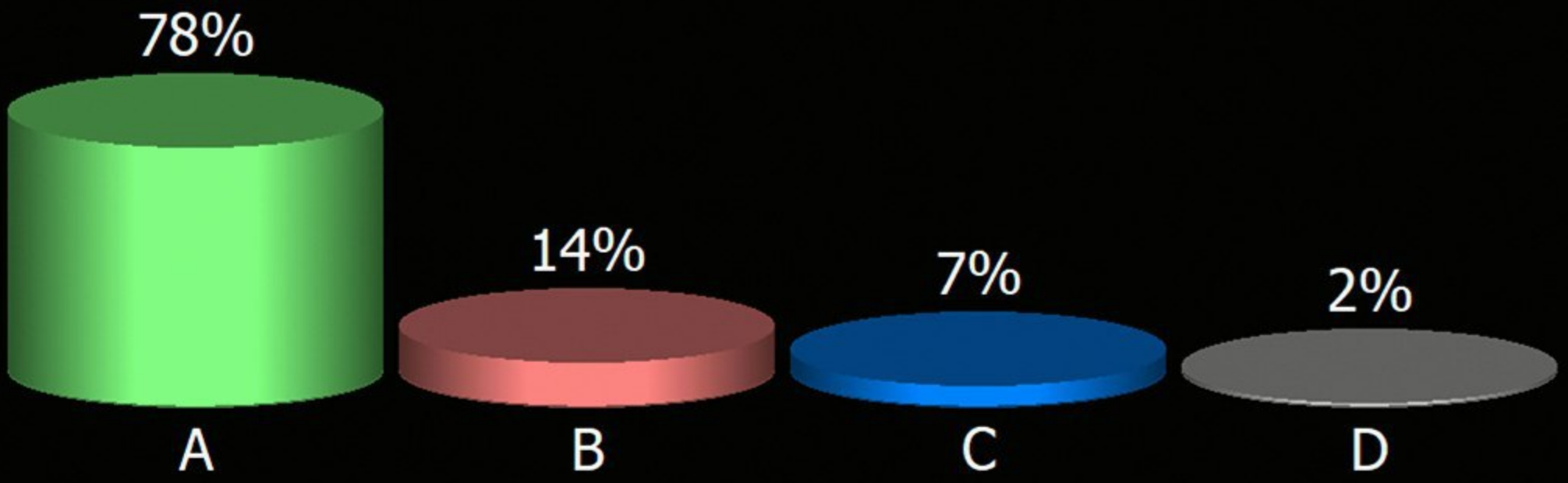

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10. TANINIZ EDİR?

a. HEMANJİOM
b. HEPATOSELÜLER ADENOM
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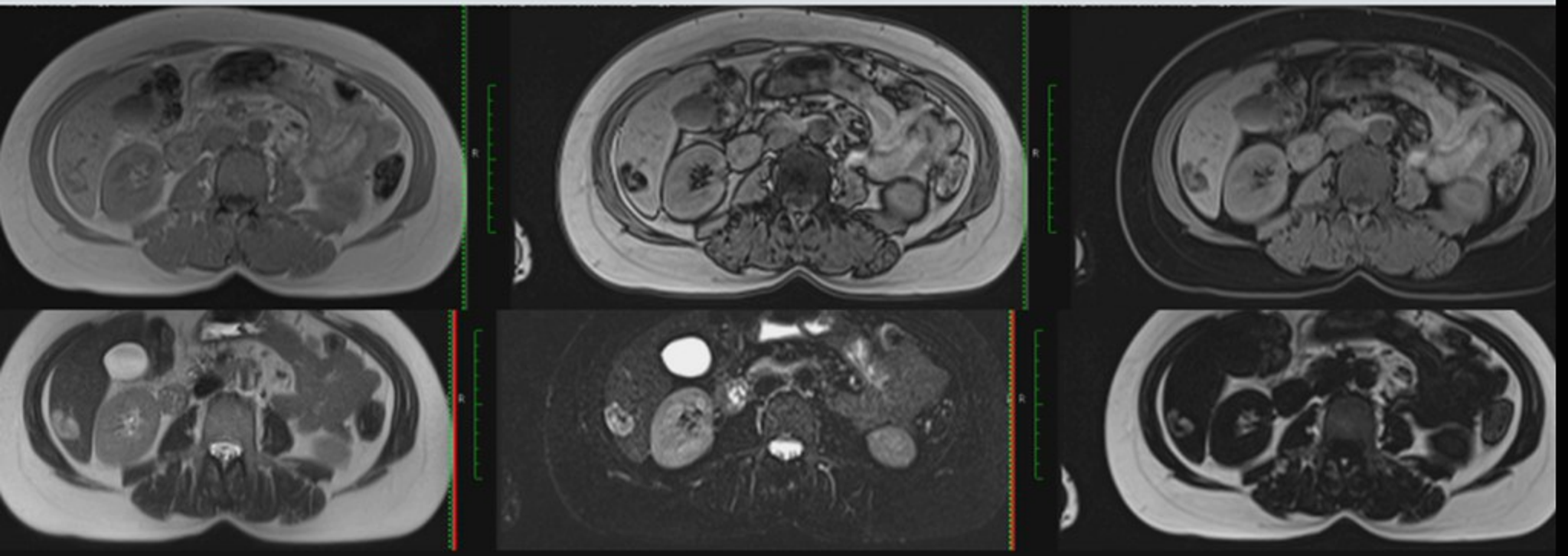

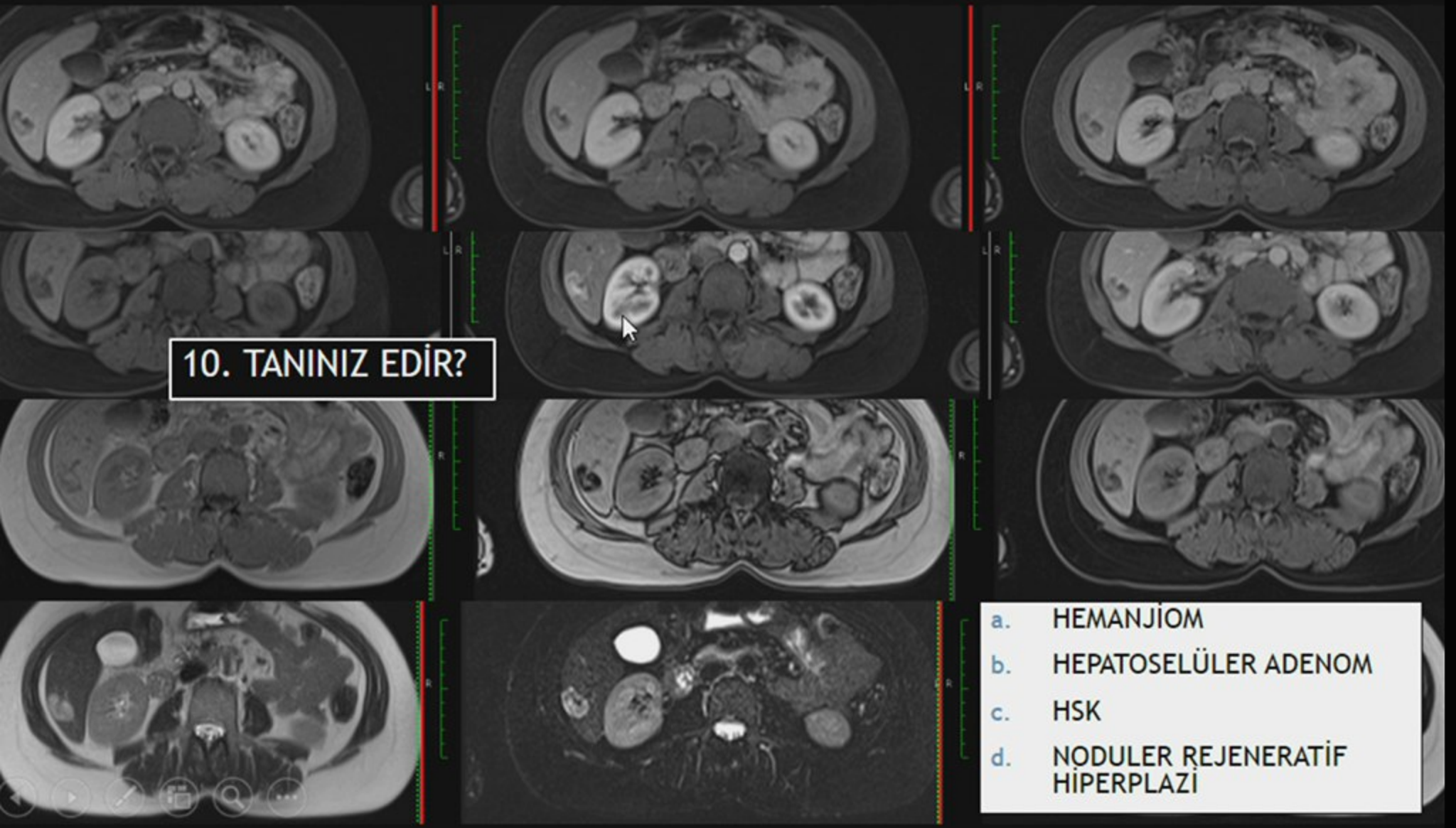
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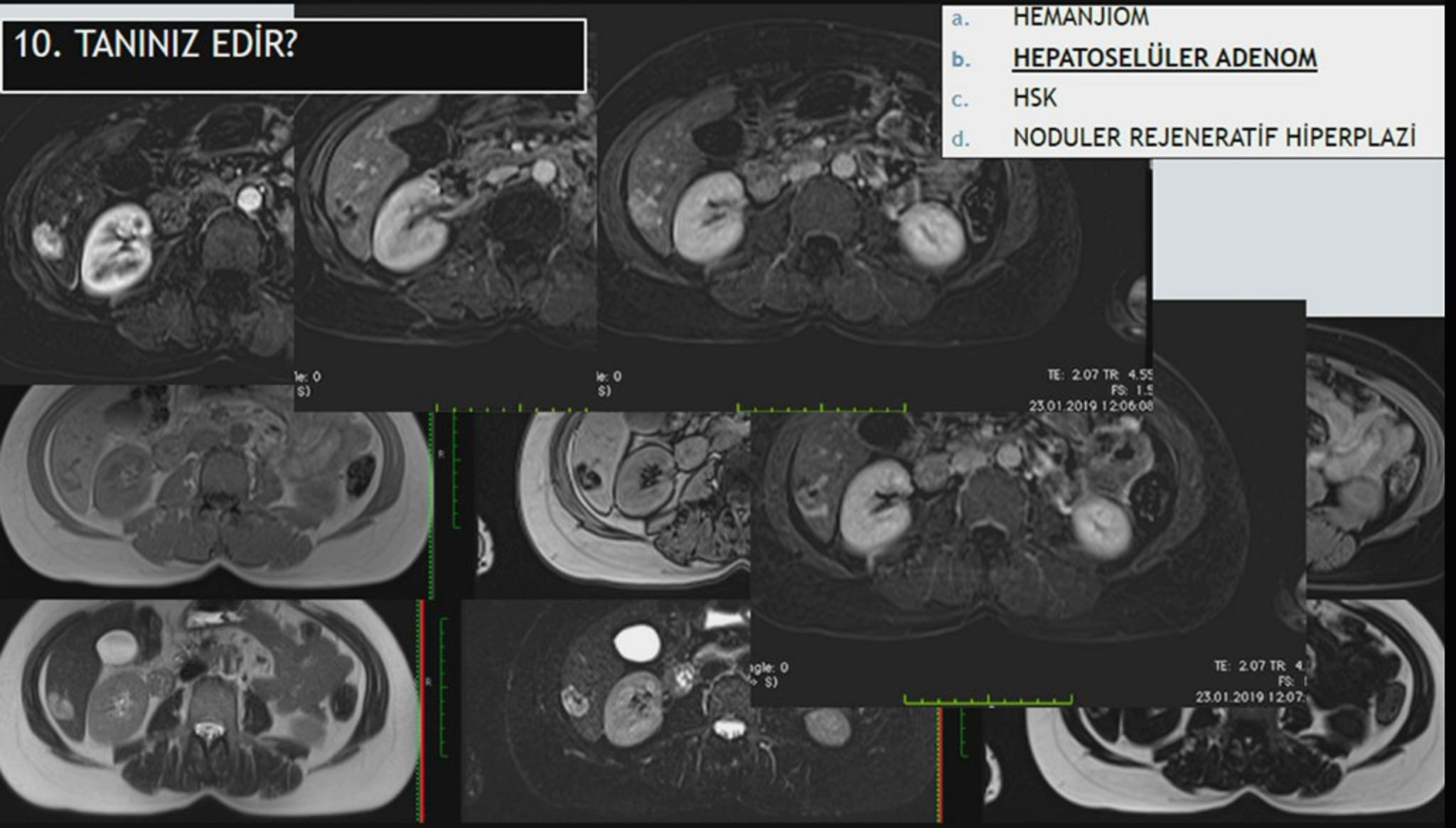

11. TANINIZ NEDİR?

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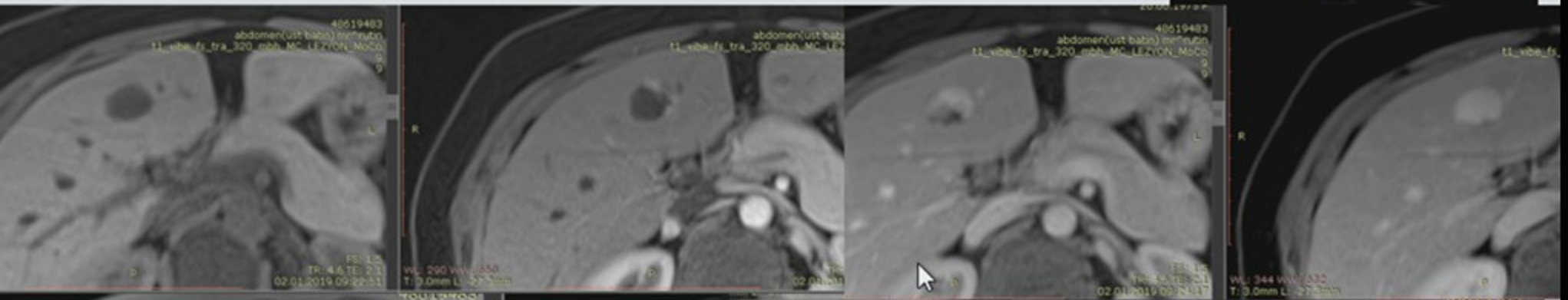
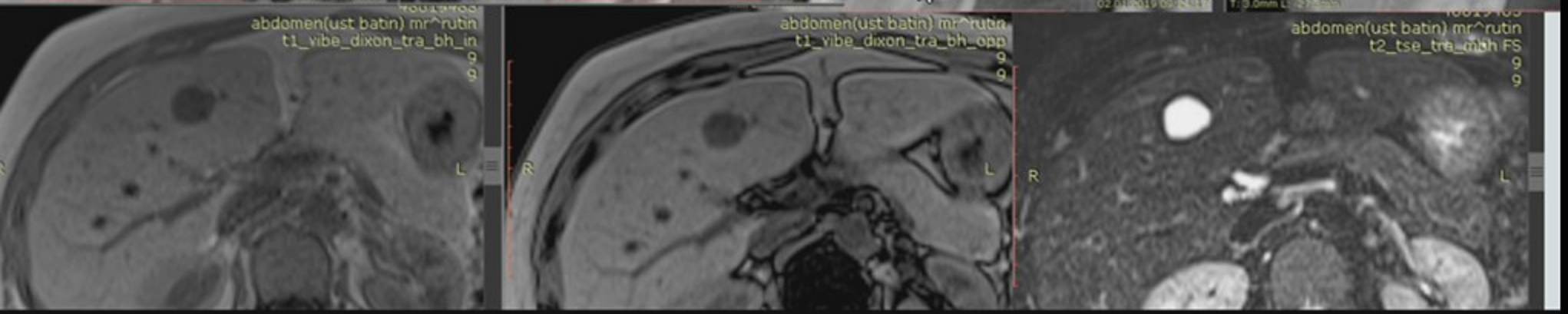
b. HEPATOSELÜLER ADENOM
c. HSK
d. NODULER REJENERATİF HİPERPLAZİ





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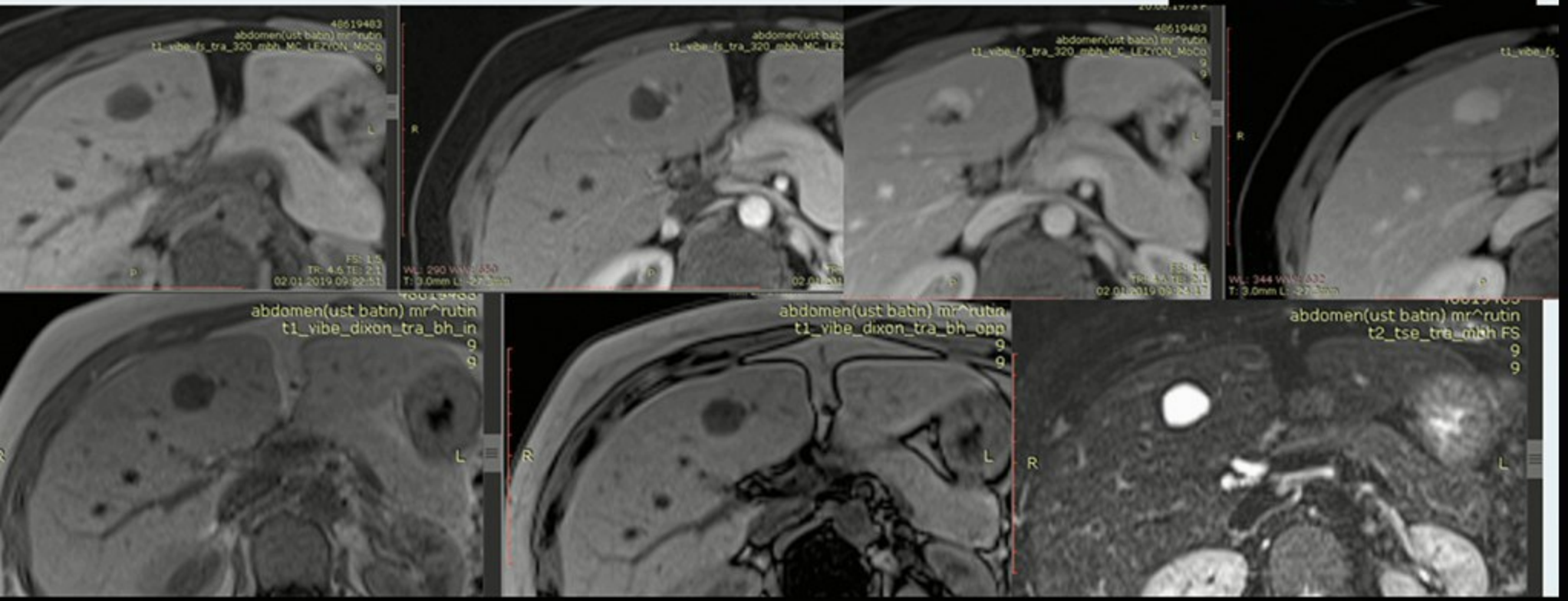

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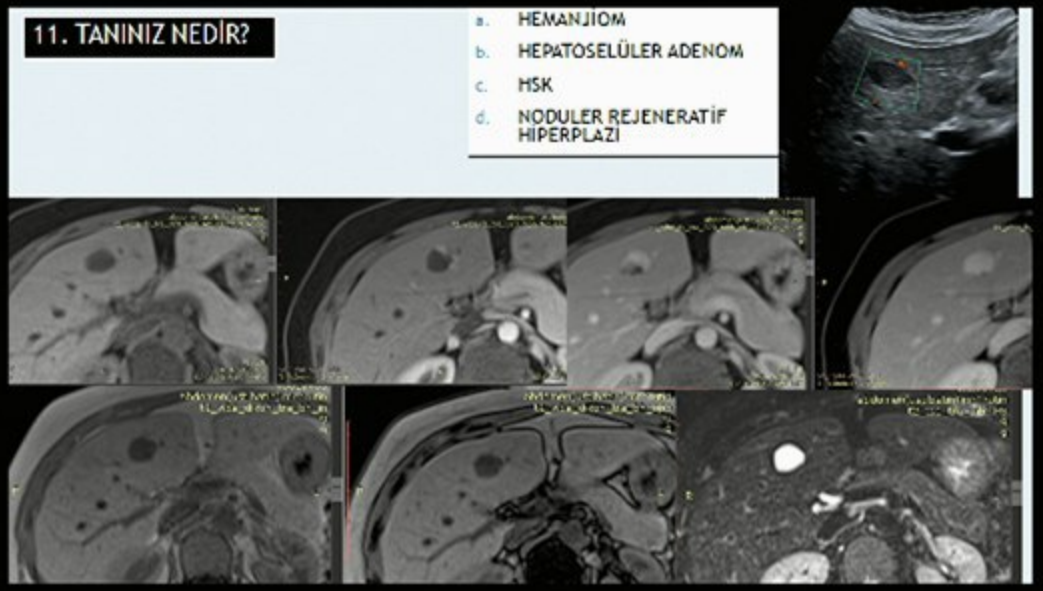

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Soru: 11

11. TANINIZ NEDİR?

a. HEMANJİOM
b. HEPATOSELÜLER ADENOM
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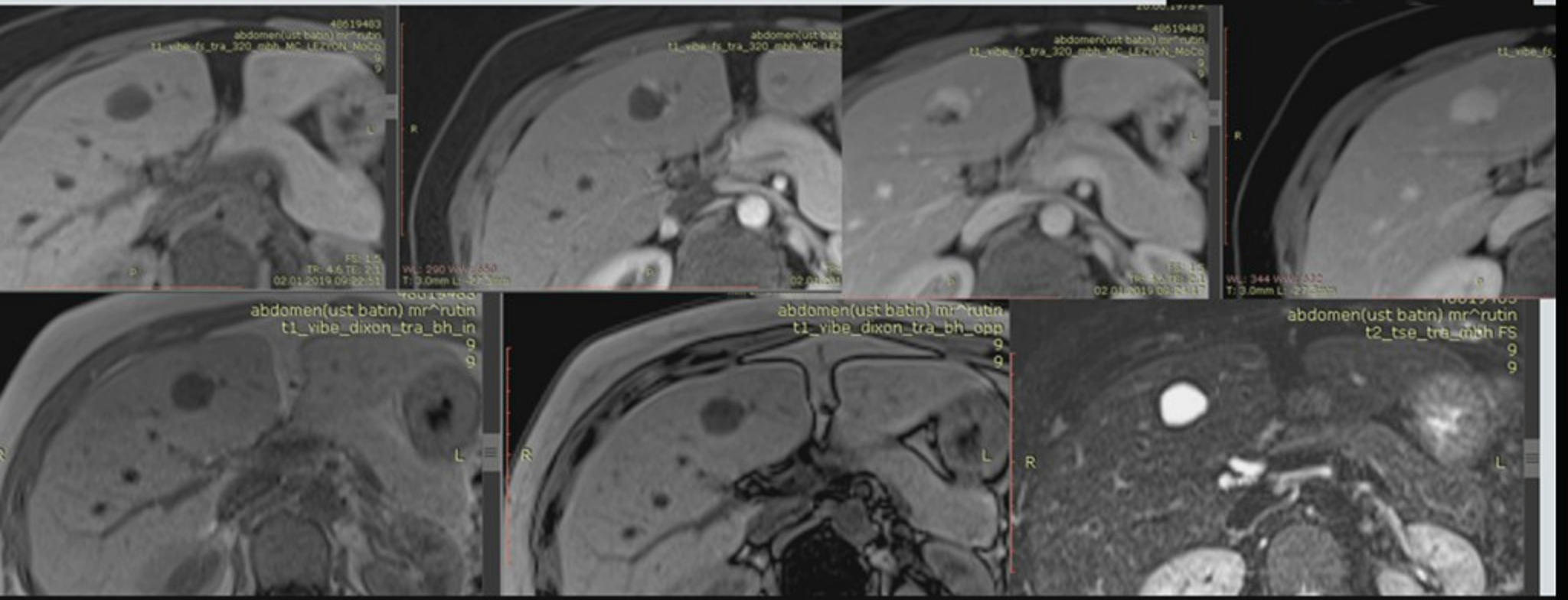

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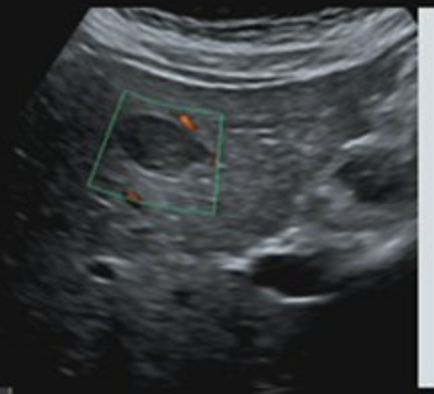

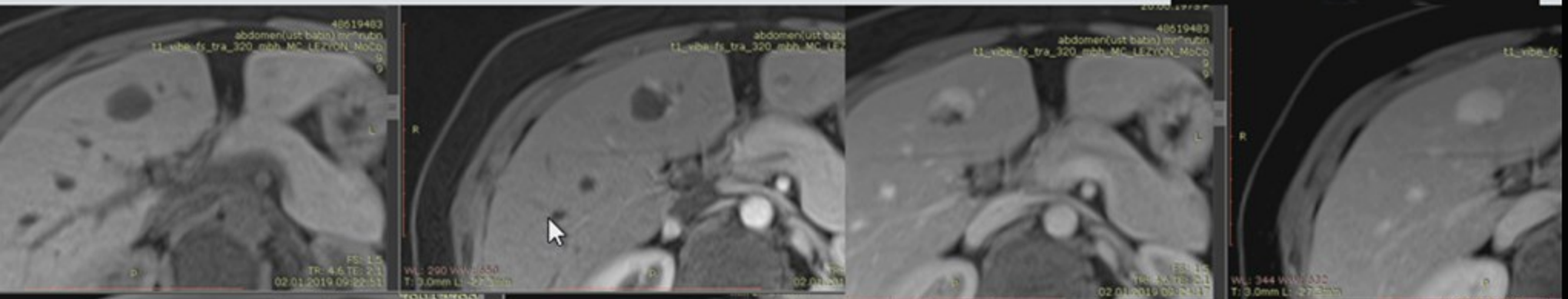

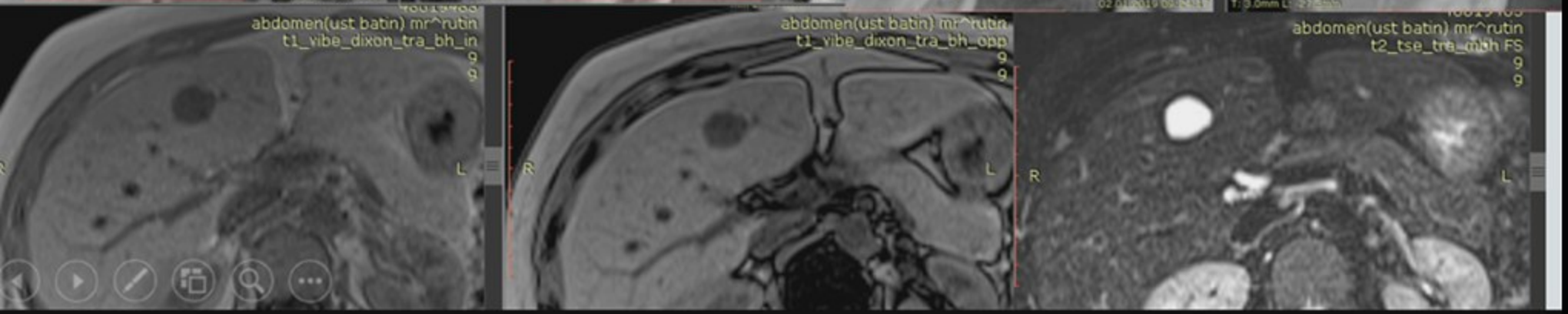

12. TANINIZ NEDİR?

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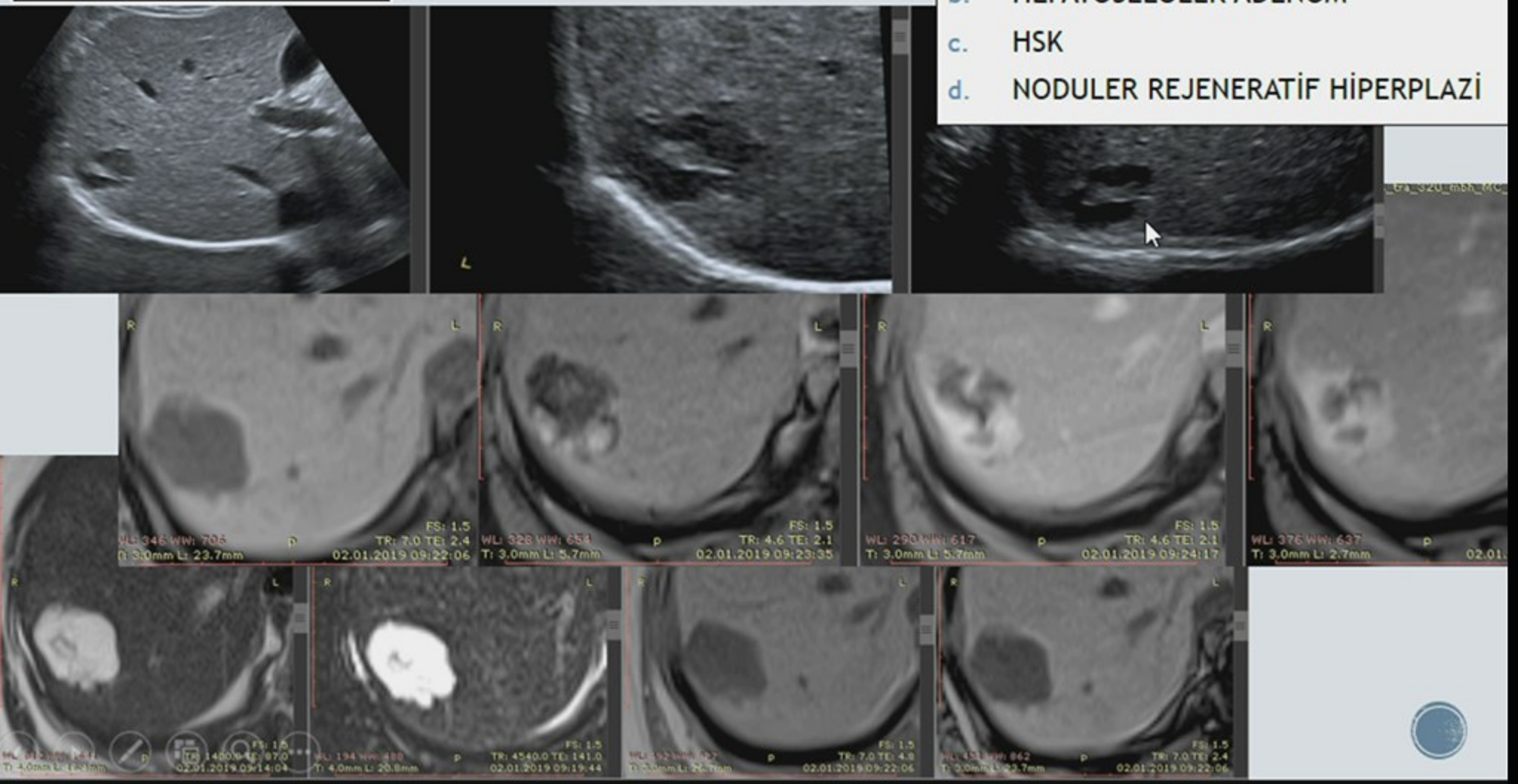

12. TANINIZ NEDİR?

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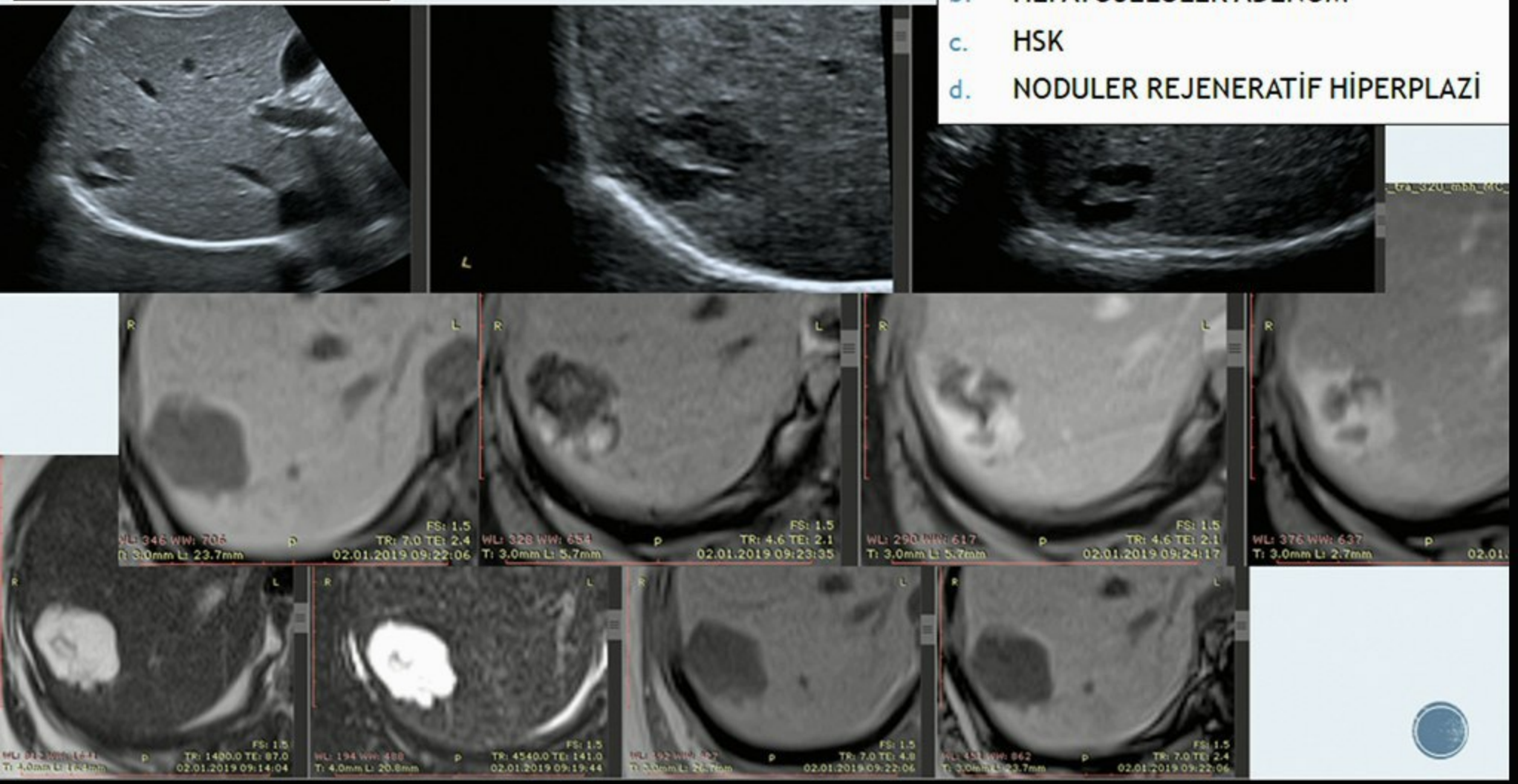
c. HSK
d. NODULER REJENERATİF HİPERPLAZİ



12. TANINIZ NEDİR?

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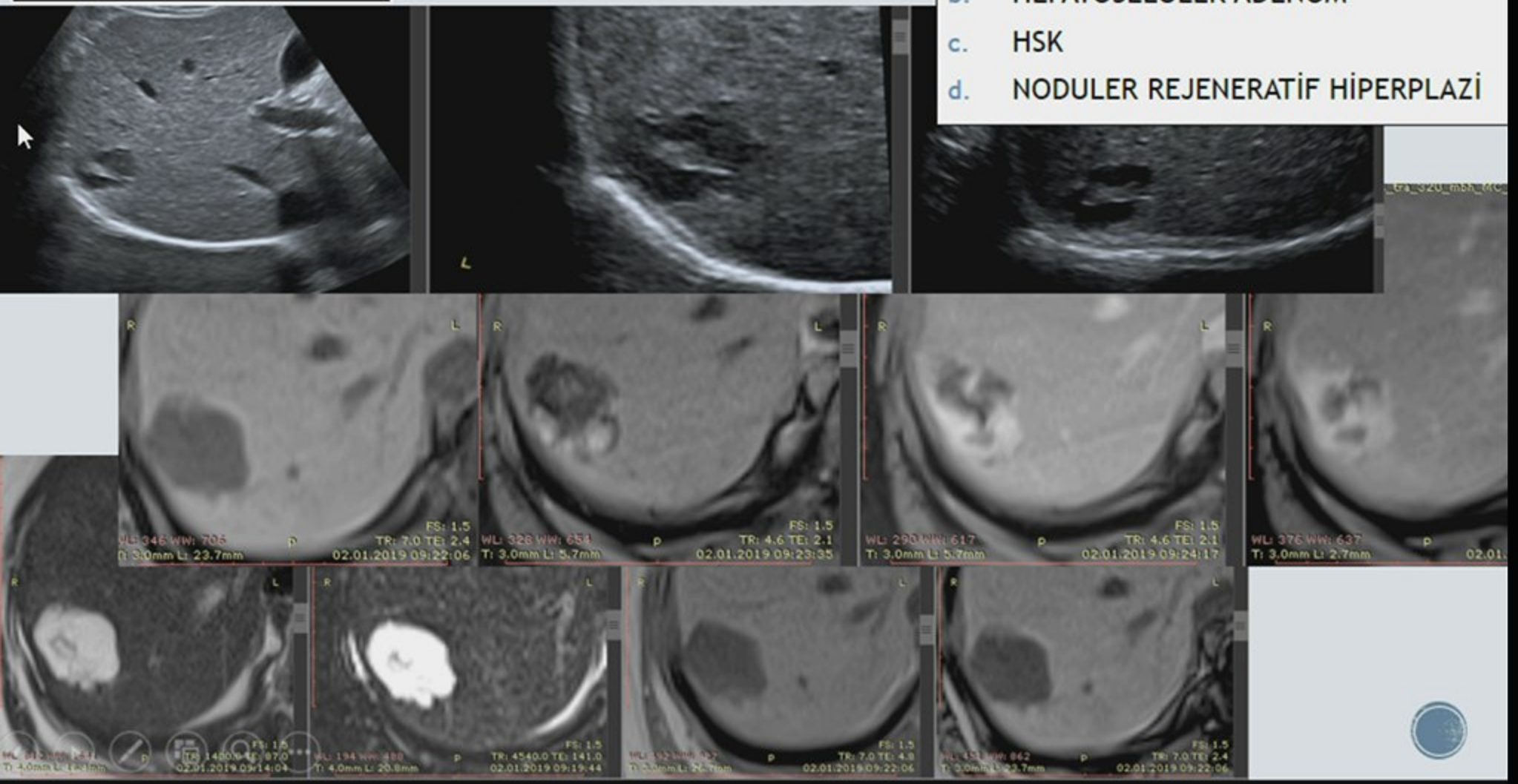


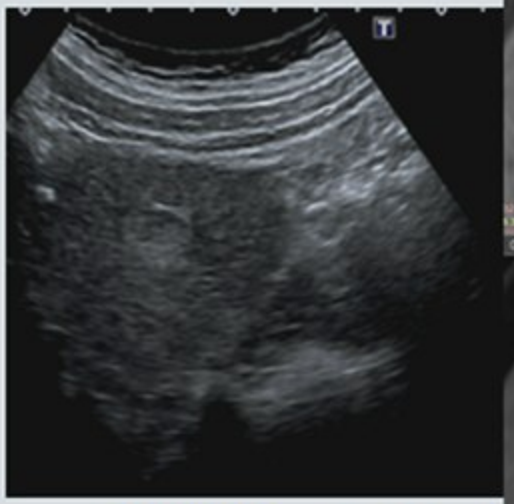
HEMANJİOM

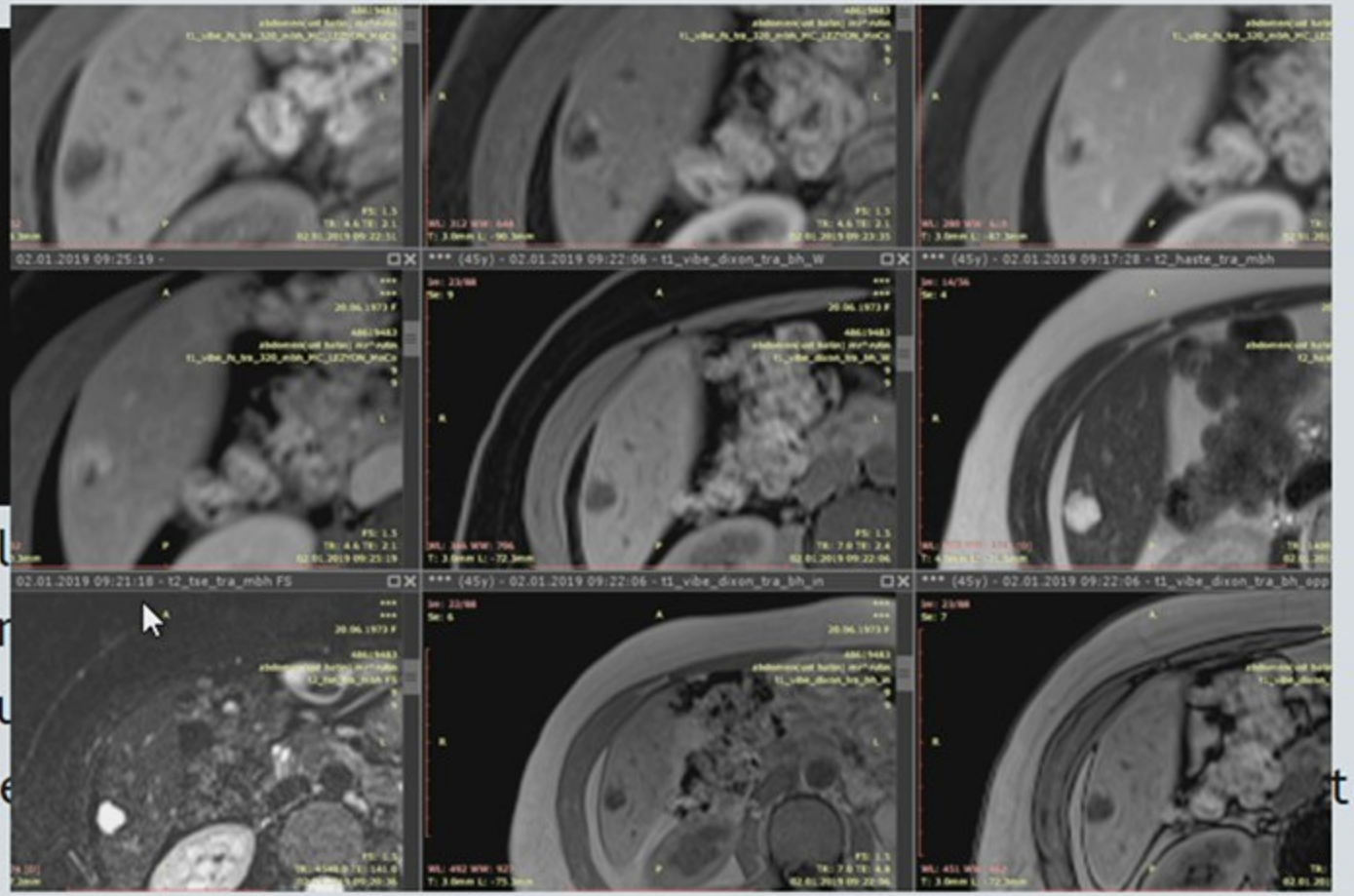
- En sık B kc lezyonu, genel populasyonun %20
- Asemptomatik
- Değişik boyutlarda damarsal kaviteler içerir
- Özellikle arka plan kc değişikliklerine bağlı tipik özellikler göstermeyebilir →saptama ve karekterizasyonda %84 sens ve %100 spf
- %40 lezyonda büyüme izlenebilir (2mm/yıl ve volümün %17/yıl)
- Histolojik olarak 3 tip: kavernöz, kapiller ve sklerozan

Kavernöz Hemanjiom

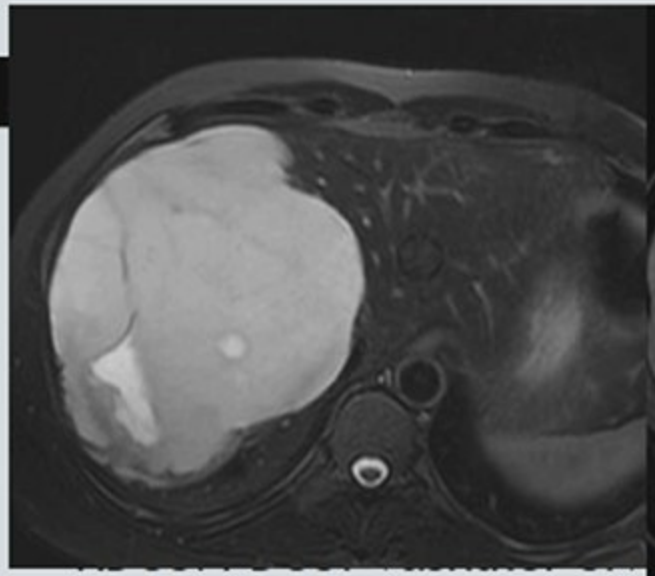
- En sık tip.
- gn.de <3cm, büyük vaskuler boşluklu
- US: hiperekoik, homojen, keskin konturlu, gn.de akustik güçlenme
- Büyük vaskuler boşluk içinde yavaş kan akımı gösterir
- RDUS/PDUS: vaskuler SN içermez
- BT:Arka plan kc N ve <3cm → izodens
- BT/MR: perfieral noduler KT(+), sentripedal, PV ve geç fazda KT ile dolar
- Dev: >5cm → santral hipodens/hipointens ile skara benzer
- Ama KT tutulumu vaskuler ile benzer
- MR: yüksek su içerik ile T2ag yüksek SN ve >120msn de hiperintensitesi sabit kalır

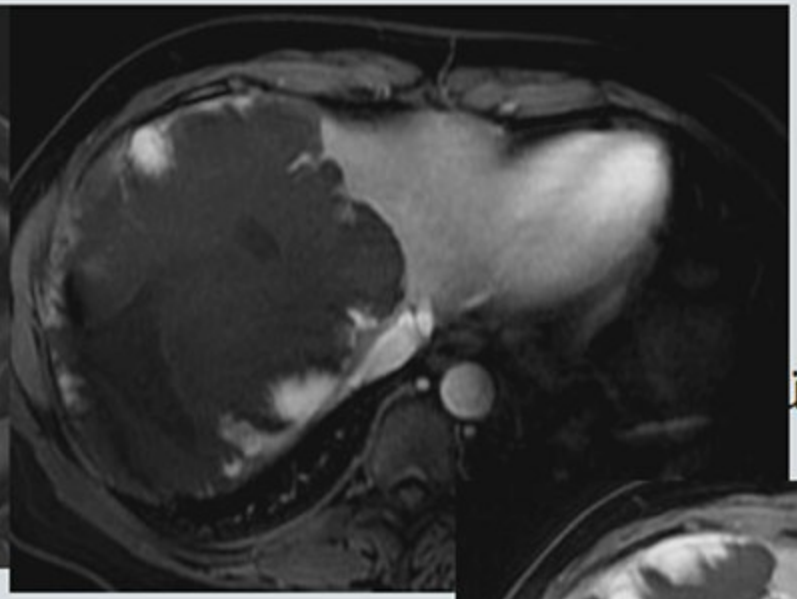
Kavernöz Hemanjiom

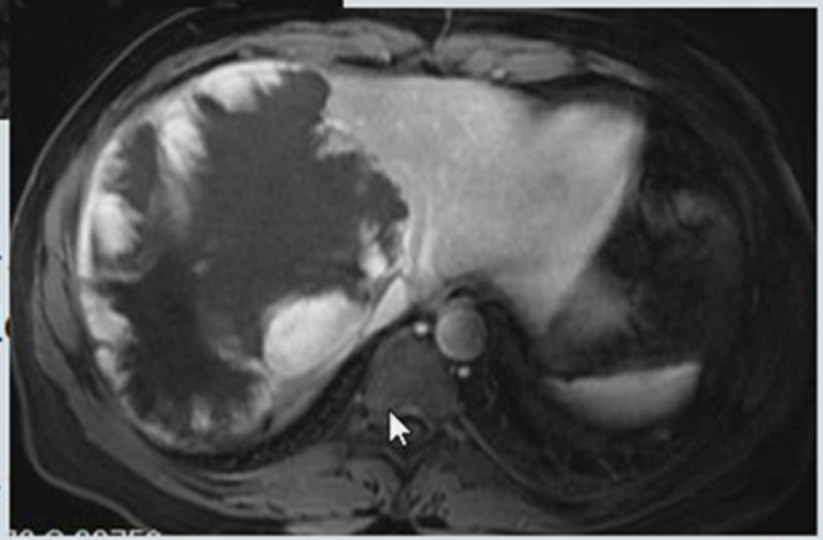




- BT/MR: perfieral
- Dev: >5cm → sar
- Ama KT tutulumu
- MR: yüksek su içe t kalır





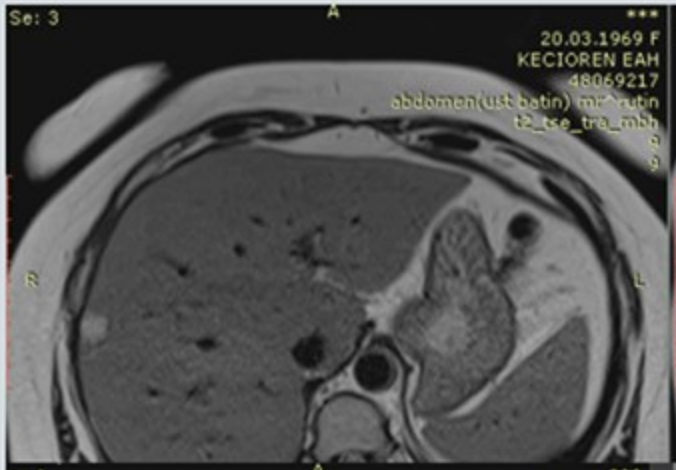


...içlenme

- BT:Arka plan kc N ve <3cm → izodens
- BT/MR: perfieral noduler KT(+), sentripedal
- Dev: >5cm → santral hipodens/hipointens il
- Ama KT tutulumu vaskuler ile benzer
- MR: yüksek su içerik ile T2ag yüksek SN ve > kalır

Kapiller Hemanjiom

- Tüm hemajiomların %16 sıdır. %42 si <1cm
- US: izo-hipoekoik (hızlı kan akımı), RDUS'de kan akımı (+)
- Steatotik Kc→perilezyonel hipoekoik halka
- BT: hiperdens halka ve T1ag: perilezyonel korunmuş alan (kimyasal şift)
- BT: lezyon çevresi hipo-izo
- KM →erken, homojen ve hızlı KT (aort ile benzer, yıkanma olmaz)
- <1cm →geçici parankimal KT(+) (AP şanta bağlı)
- Flushing filling
- AT: hipervaskuler met (geç fazda hipodens iken hemanjiom hiperdens/intens)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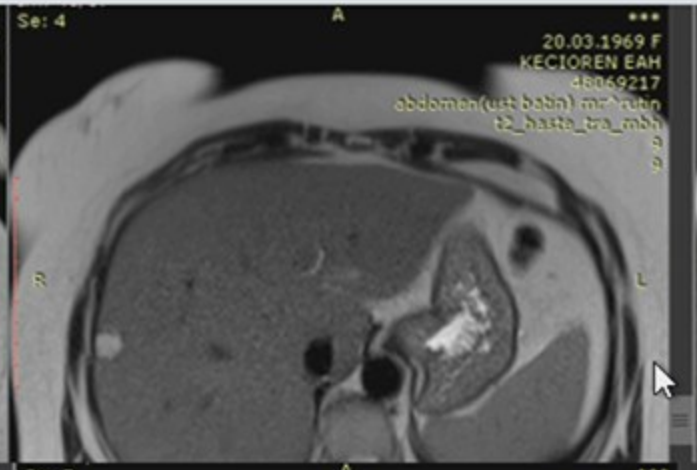
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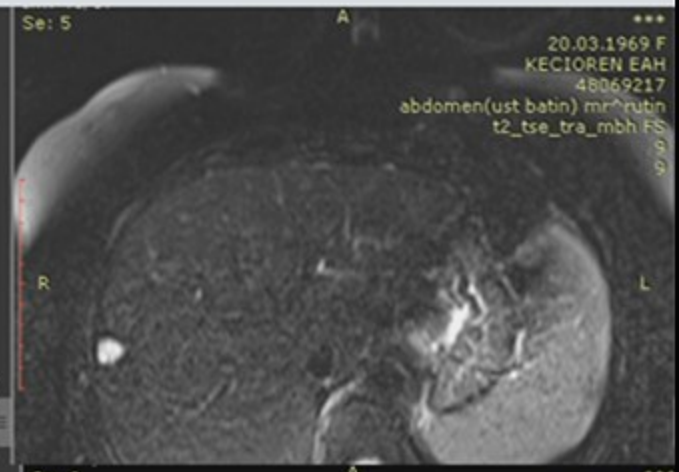
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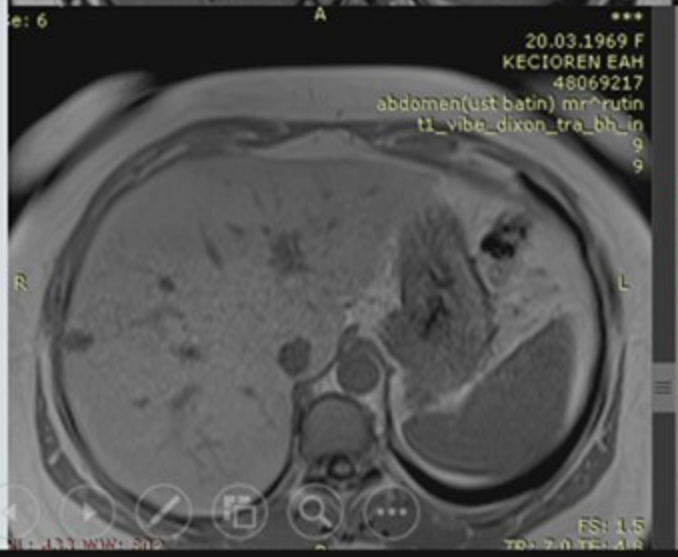
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KECIOREN EAH
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abdomen(ust batin) mr^rutin
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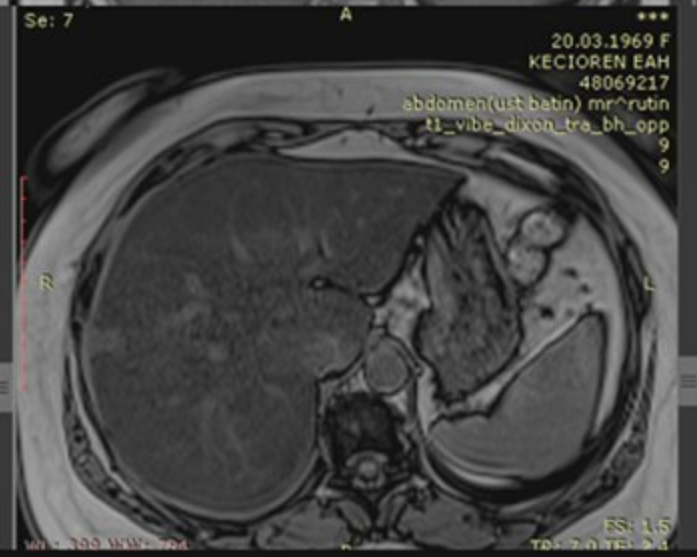
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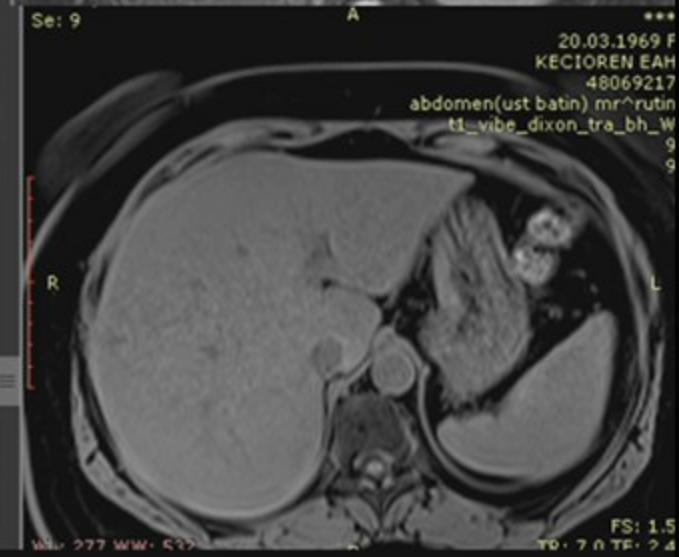
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48069217
abdomen(ust batin) mr^rutin
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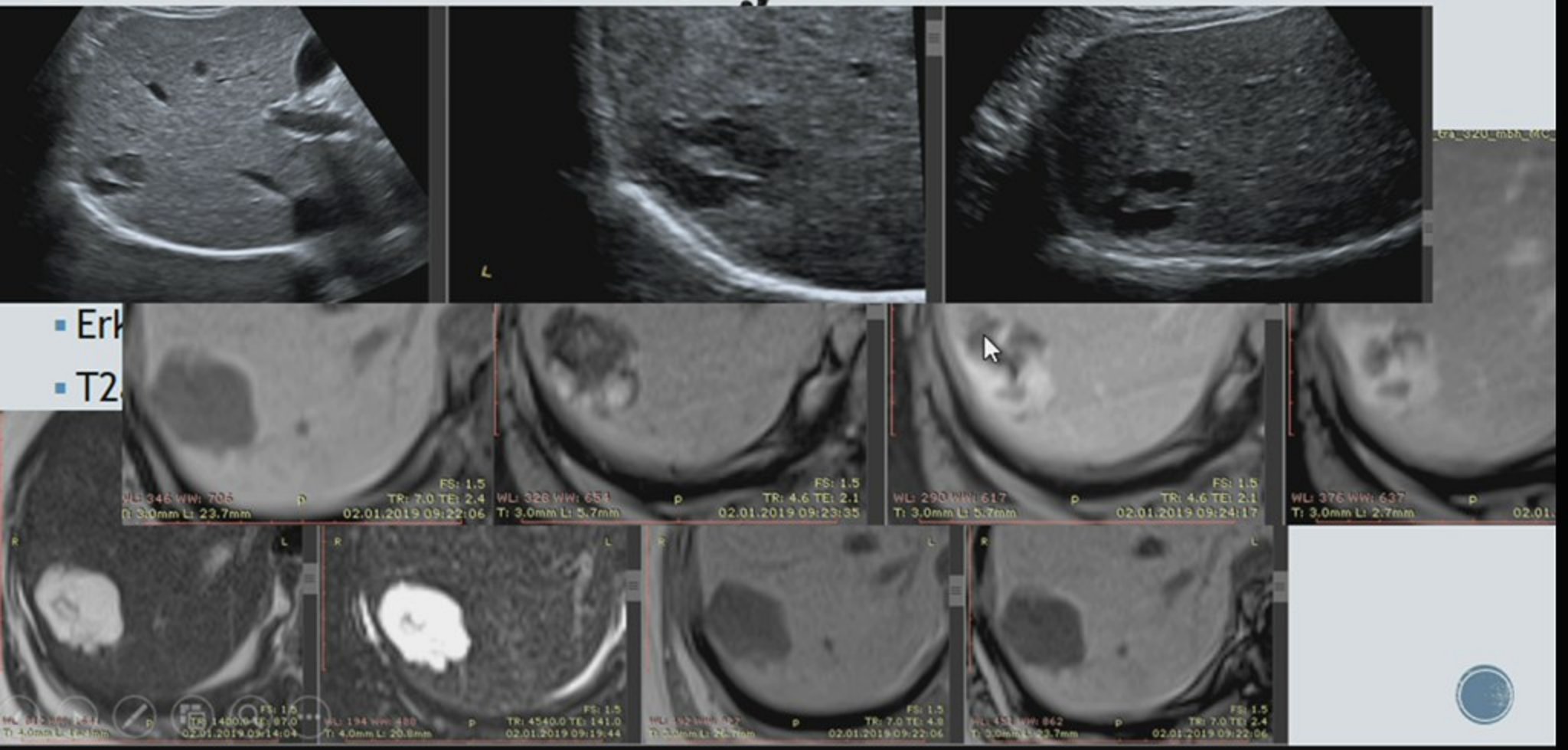
R
L
FS: 1.5

Sklerozan Hemanjiom

- Yoğun fibrozis (santralden başlar) ve vaskuler boşlukları doldurur.
- US, BT ve MR değişken
- Hemorajik, kistik dejenerasyon veya fibrozise bağlı heterojenite içerir
- Preop tanı zor
- Erken KT olmayabilir, inhomojen ve progresif KT tutulumu olabilir
- T2ag de hafif hiperintens(gn.de periferal)
- AT: kolanjio, met

Sklerozan Hemanjiom



	Capillary haemangioma	Cavernous haemangioma	Sclerosed haemangioma
Histological composition	Reduced vascular spaces Extensive connective tissue	Large vascular spaces Not very extensive connective tissue	Extensive beginning fibrosis at the centre of the lesion
Size	Small size (in general < 1 cm)	Lesion < 3 cm = typical appearance Giant > 4 cm	Average size (3.7 cm on the average)
Morphology	Nodular, homogenous	Well defined, internal septa	Geography map appearance, central scar, capsular retraction, punctiform calcifications
Echogenicity	Hypoechogenic, homogenous Posterior enhancement	Hyperechogenic, homogenous Posterior enhancement	Heterogeneous, hypoechogenic centre
MRI: T2-weighted images	Hyperintense, homogenous	Hyperintense, homogenous	Heterogeneous, peripheral hyperintense, central hypointense
Kinetics of the contrast enhancement	Rapid flow, early enhancement, intense, in flash, persistent at late time	Slow flow, nodular peripheral enhancement, progressive and complete centripetal filling	Slow flow, peripheral nodular enhancement, very late homogeneous filling (3 h)
Perilesional environment	Transitory zone of perilesional enhancement, arterioportal shunt		Enhancement in perilesional ring, capsular retraction

TEŞEKKÜRLER