B mode renal US: Normal and pathological findings

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Content of presentation

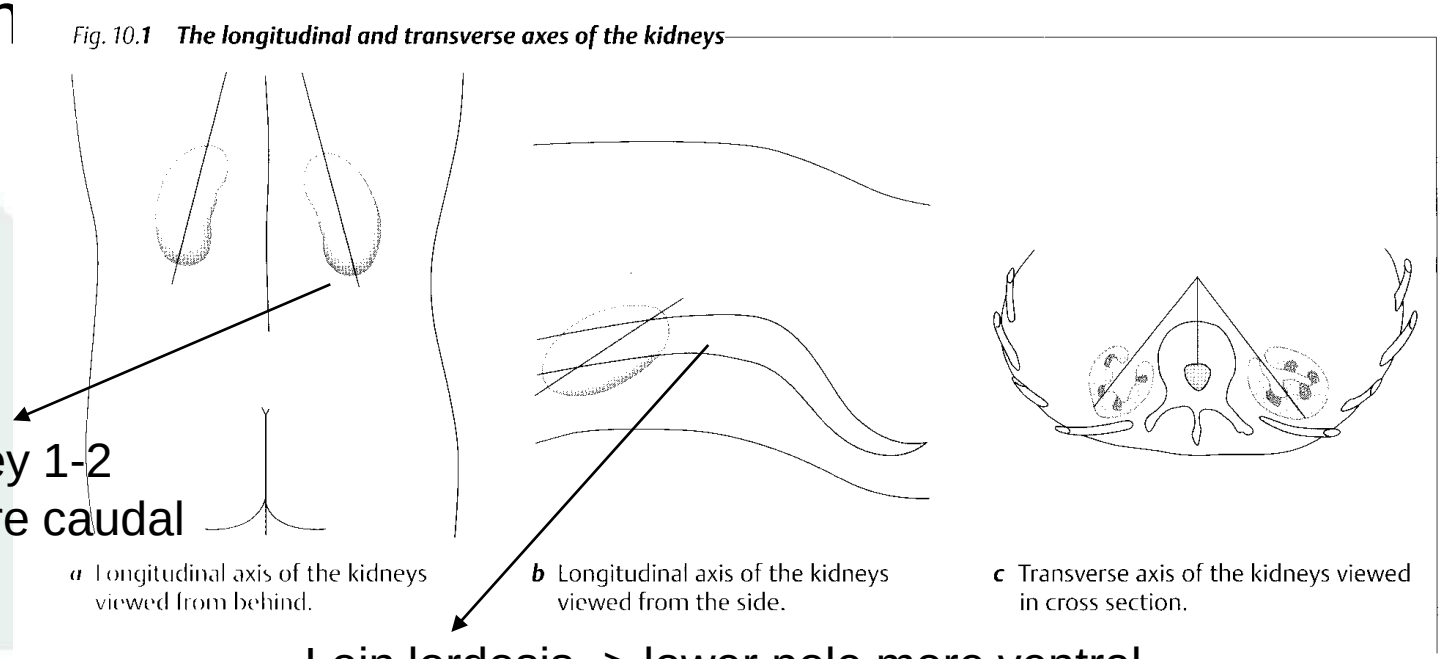
- **Normal renal echo anatomy**
- **(Normal) anatomical form variants**
- **Renal echo pathology :**

➢ Renoparenchymatous diseases

➢ Focal lesions:
- lithiasis (hydronephrosis)
- cysts
- tumours

Kidney location and orientation

- Bean shaped (lateral convex and medial concave)
- Located retroperitoneally
- On both sides of the vertebral column
- Sliding on the m quadratus lumborum and psoas m



R kidney 1-2 cm more caudal than L

Loin lordosis -> lower pole more ventral

Visualisation of right kidney

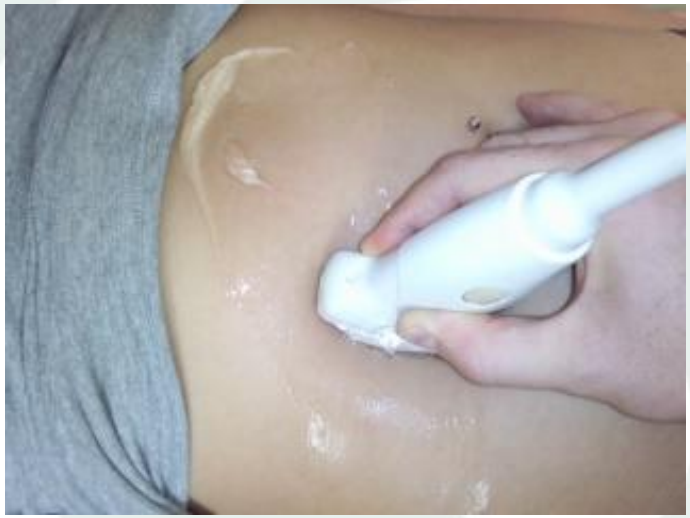
- Patient on his back
- Sometimes better visualisation in left lateral position with R arm behind head
- Ask to take a deep breath (to see upper pole)
- Liver = acoustic window
- 2 approaches :

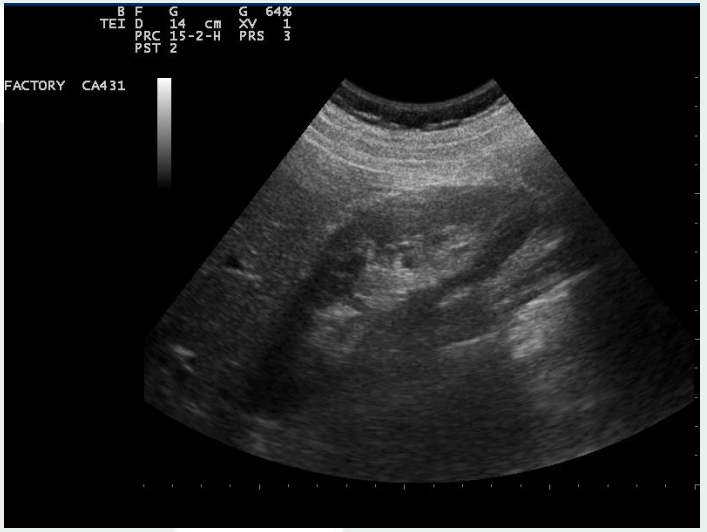
 - anterior axillary line (slim patients)

 - scan subcostally , at dorsolateral side

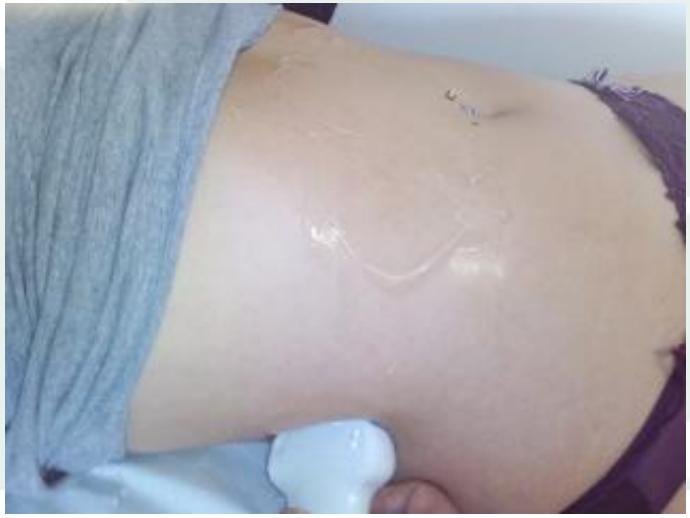
Visualisation right kidney

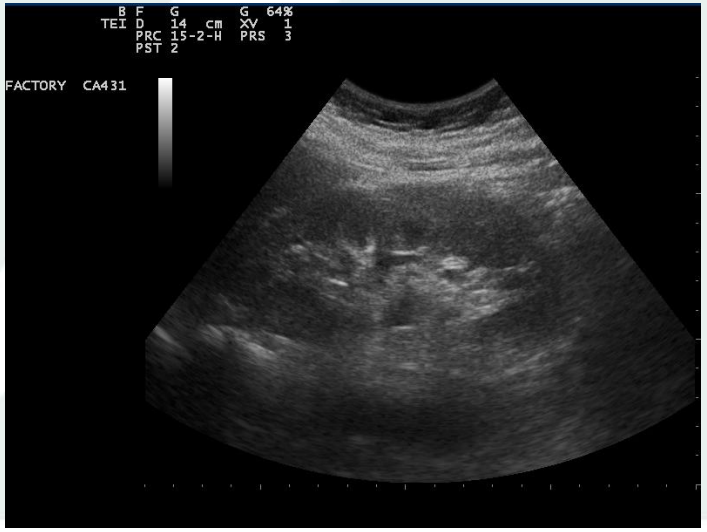
Ventrolateral





Posterolateraal

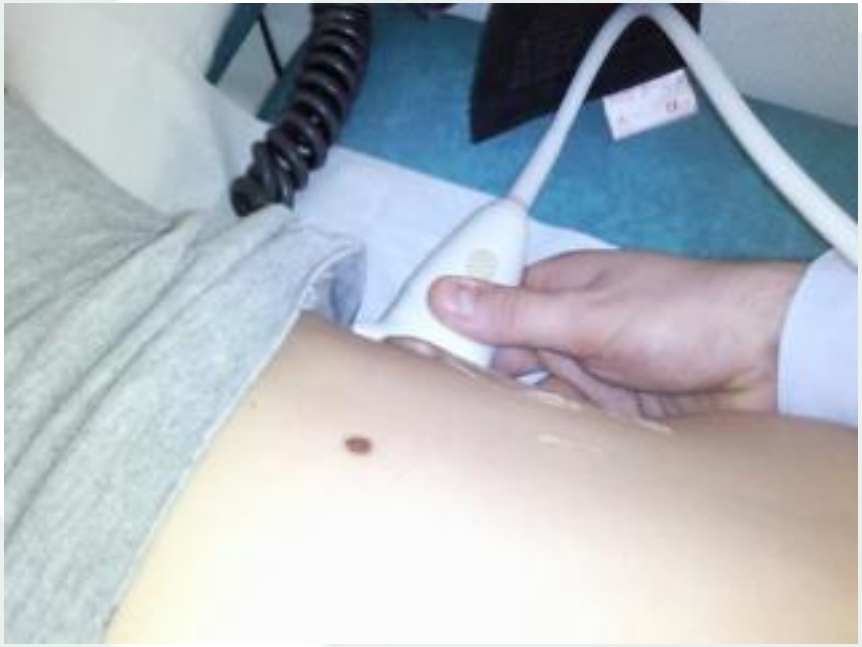


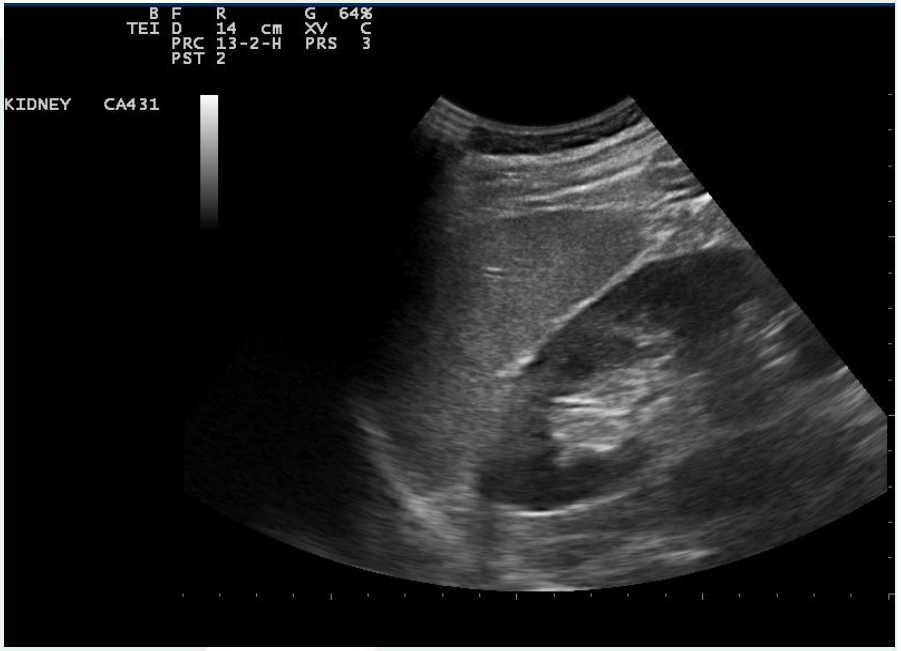


Visualisation of left kidney

- Often harder than R kidney
- Ptn on his back (or prone for biopsy)
- Or right lateral decubitus with L arm above head
- Deep inspiration often needed
- Subcostal, dorsolateral approach
- Position the TD very posterior
- Spleen = acoustic window

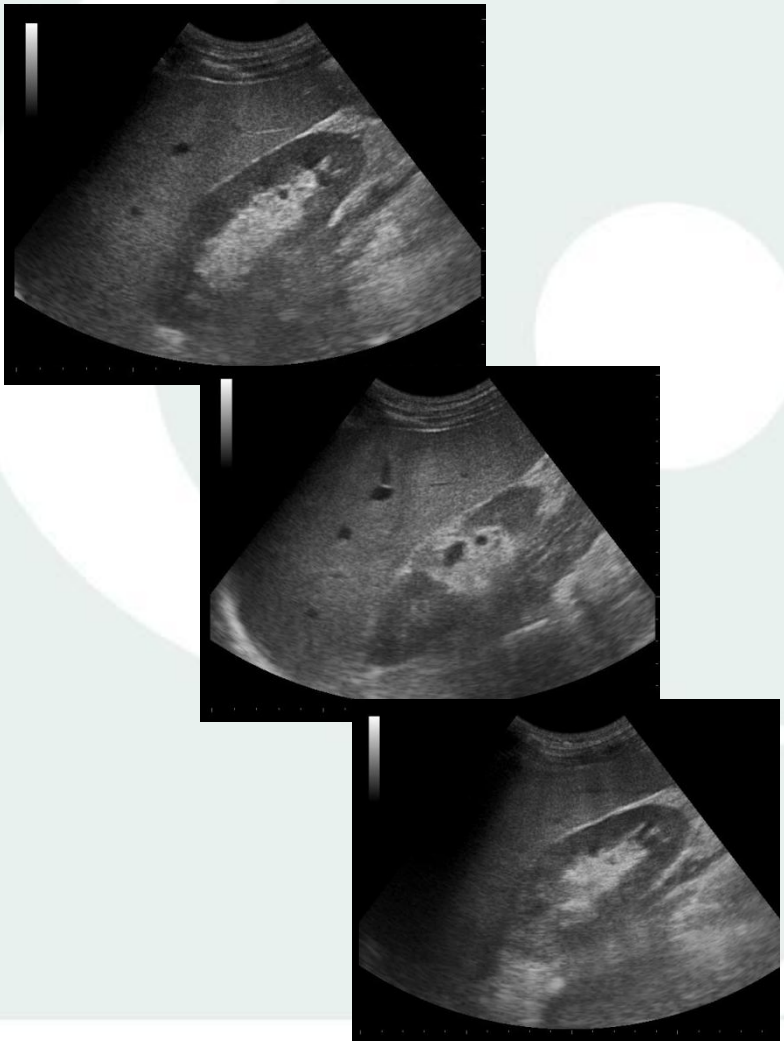
Visualisation of the left kidney



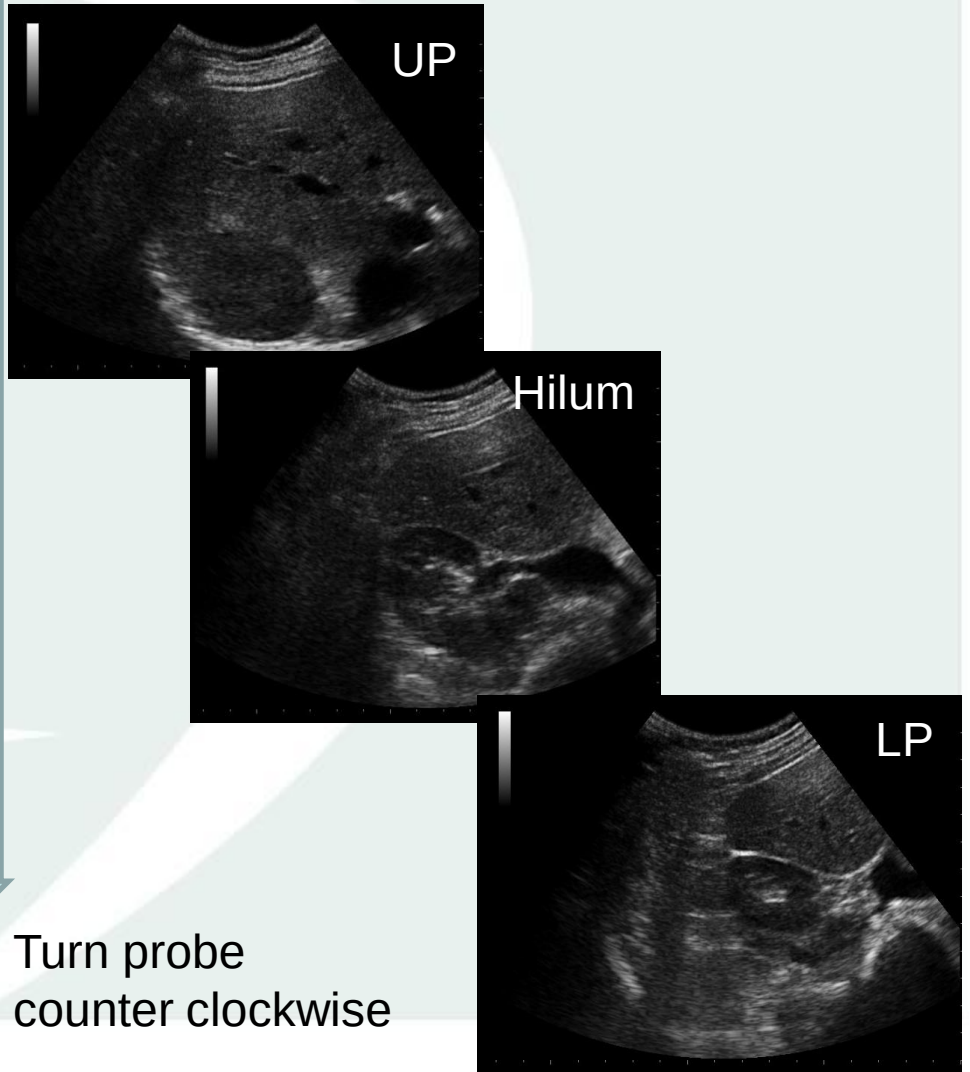


Documentation

3 longitudinal images
(lateral/middle/medial)



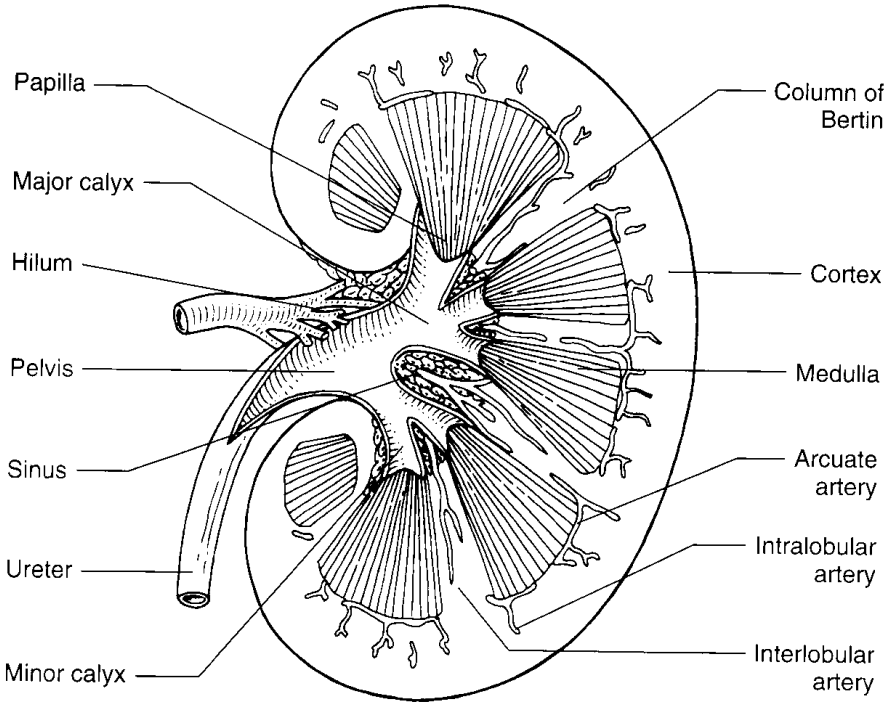
3 transverse images
(upper/middle (hilum)/lower pole)

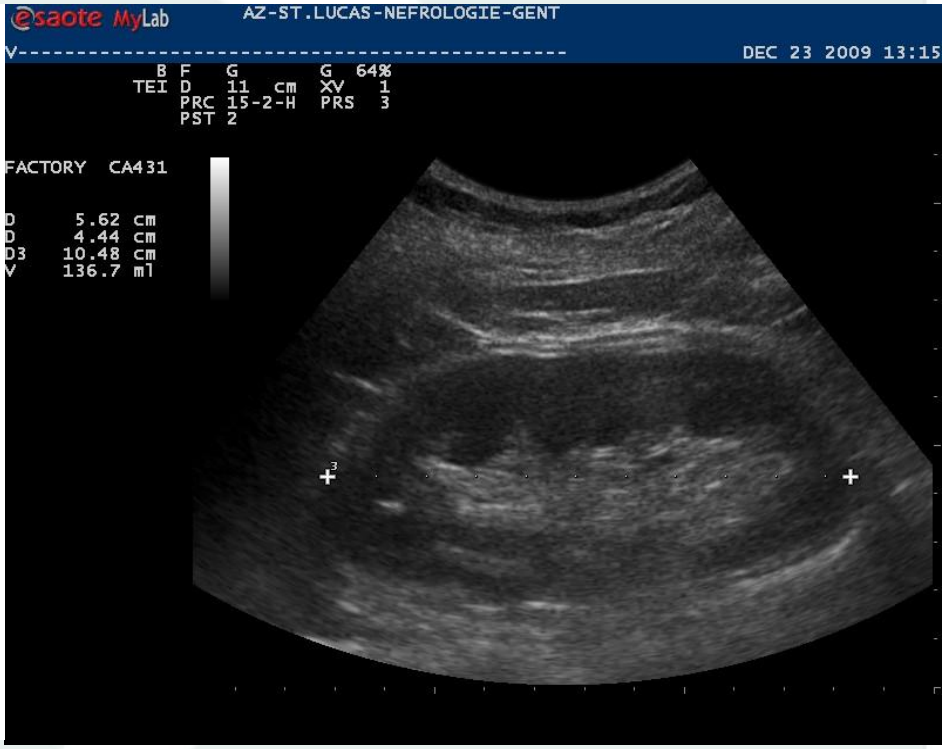


Turn probe
counter clockwise

Echo-anatomy B mode

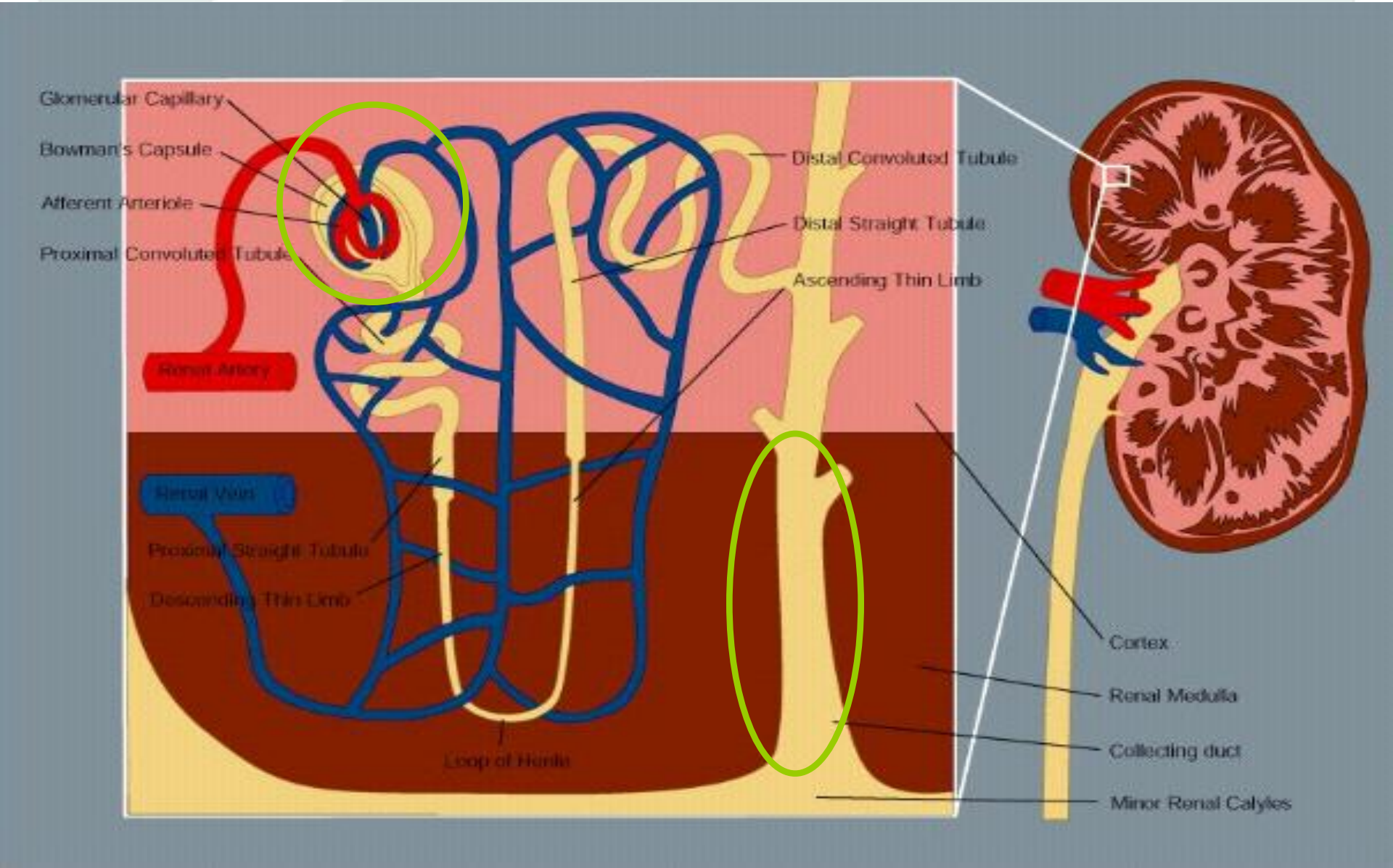
Parenchyma: cortex + medulla (8-20 pyramids)
Pelvicalicial system
Sinus
Hilus





Kidneys are enclosed by an adipose capsule (thickness varies with constitution)
Can have have high or low echogenicity

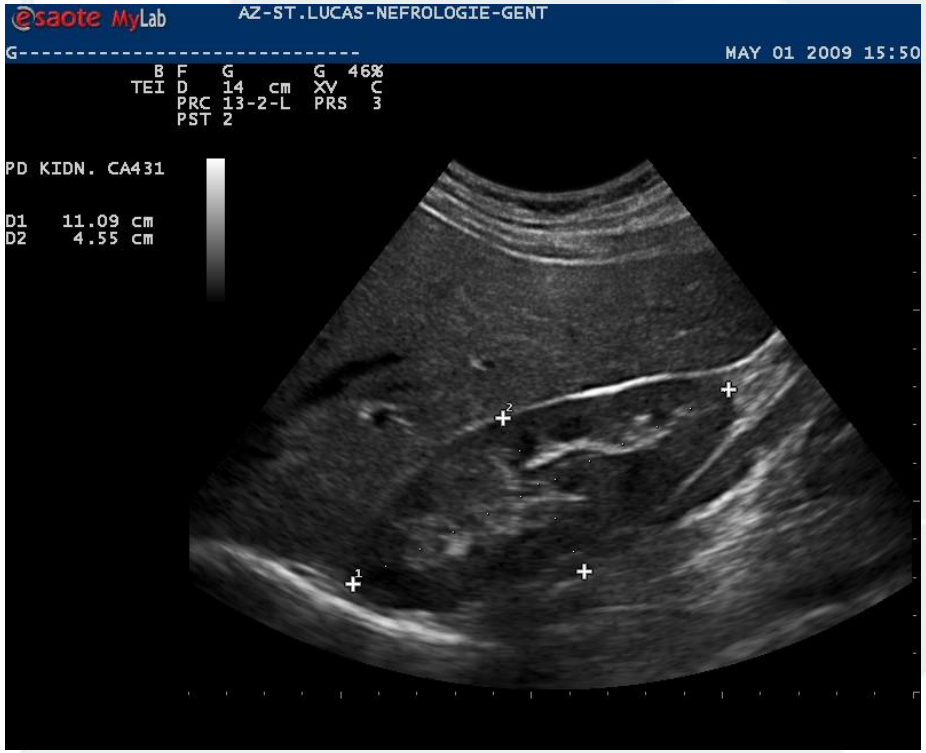
Why is medulla darker than cortex?



Kidney surface

Smooth

= normal



Uneven:

DD:

-nephrosclerosis

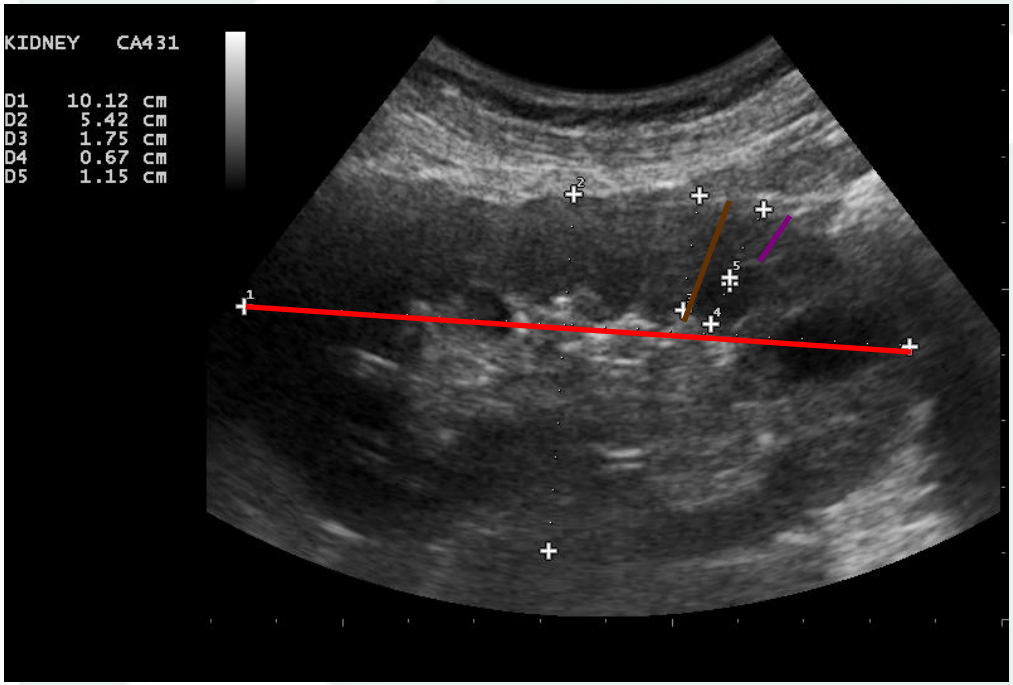
-sequel pyelonephritis

-renal infarction

-persistent fetal lobulation

- …

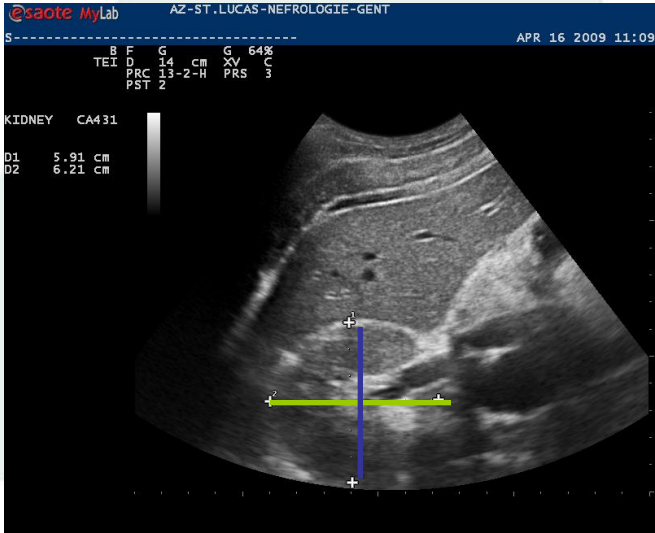
Kidney dimensions



- Length : 9-12 cm
  (L kidney slighty longer than R))
- Width: 4-7 cm
- Height: 3-5 cm
- Resp.mobility:3-7cm

Parenchymal width (cortex + medulla)
= from tip of medullary pyramid (papilla) to the kidney surface
= normal : 15 - 25 mm

Cortex width
= 8-11 mm



Parenchym-pelvis ratio

- Alternative way to judge the parenchymal width
- Ventral + dorsal parenchymal width / width of pelvis
- Normal reference values (*):

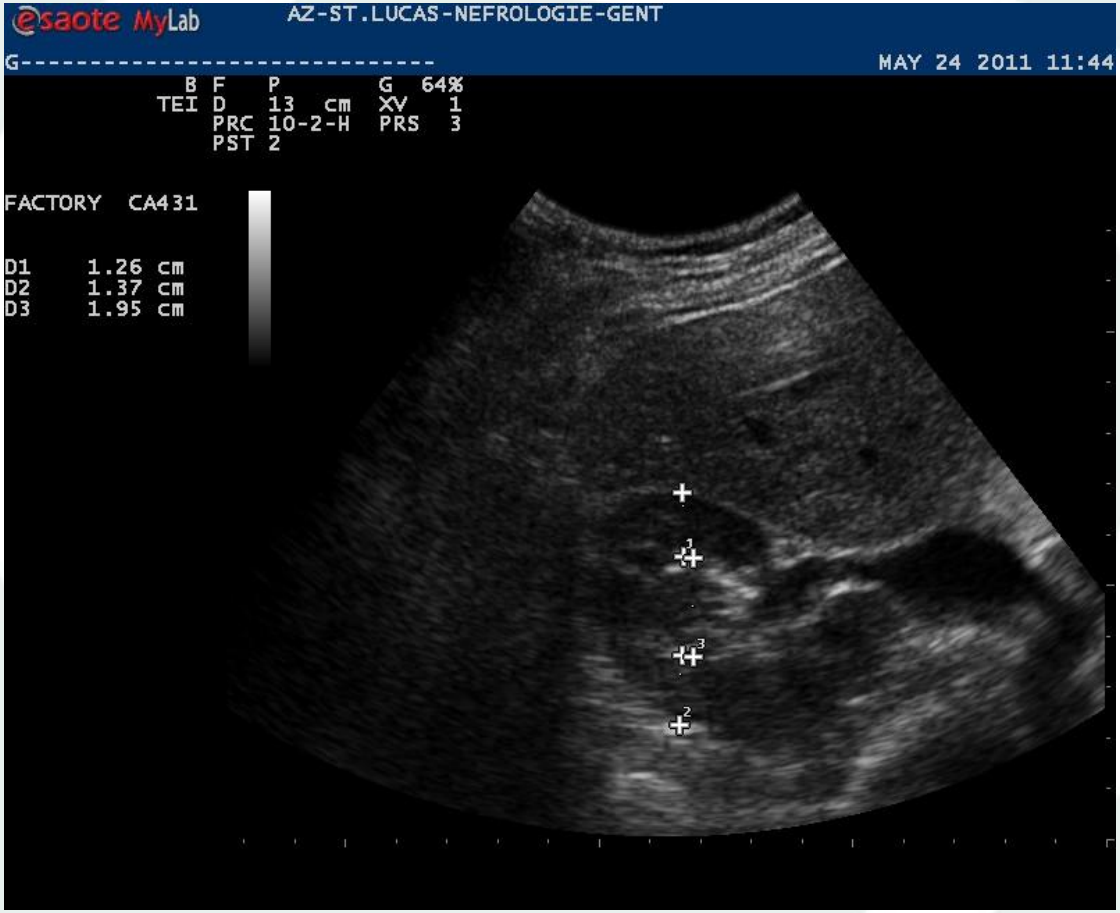
< 30 y : > 1.6

31-60 y: 1.2 – 1.6

> 60 y : 1.1

(*)Sono Grundkurs. Thieme. Matthias Hofer.

Female 35 y



1.26 + 1.37/ 1.95

=

1.4

Measure ant & post parenchymal width at level of the hilus

Do the kidneys have normal size?

Poor correlation renal length – body length

Better correlation renal volume – body weight

Normal renal size can best be estimated as renal volume (ml), which should be twice body weight (Kg) **

**J Radermacher. Urologe A. 2005 Nov;44(11):1351-63

Volume

- Formula ellipsoid:

  Length x width x hight / 2

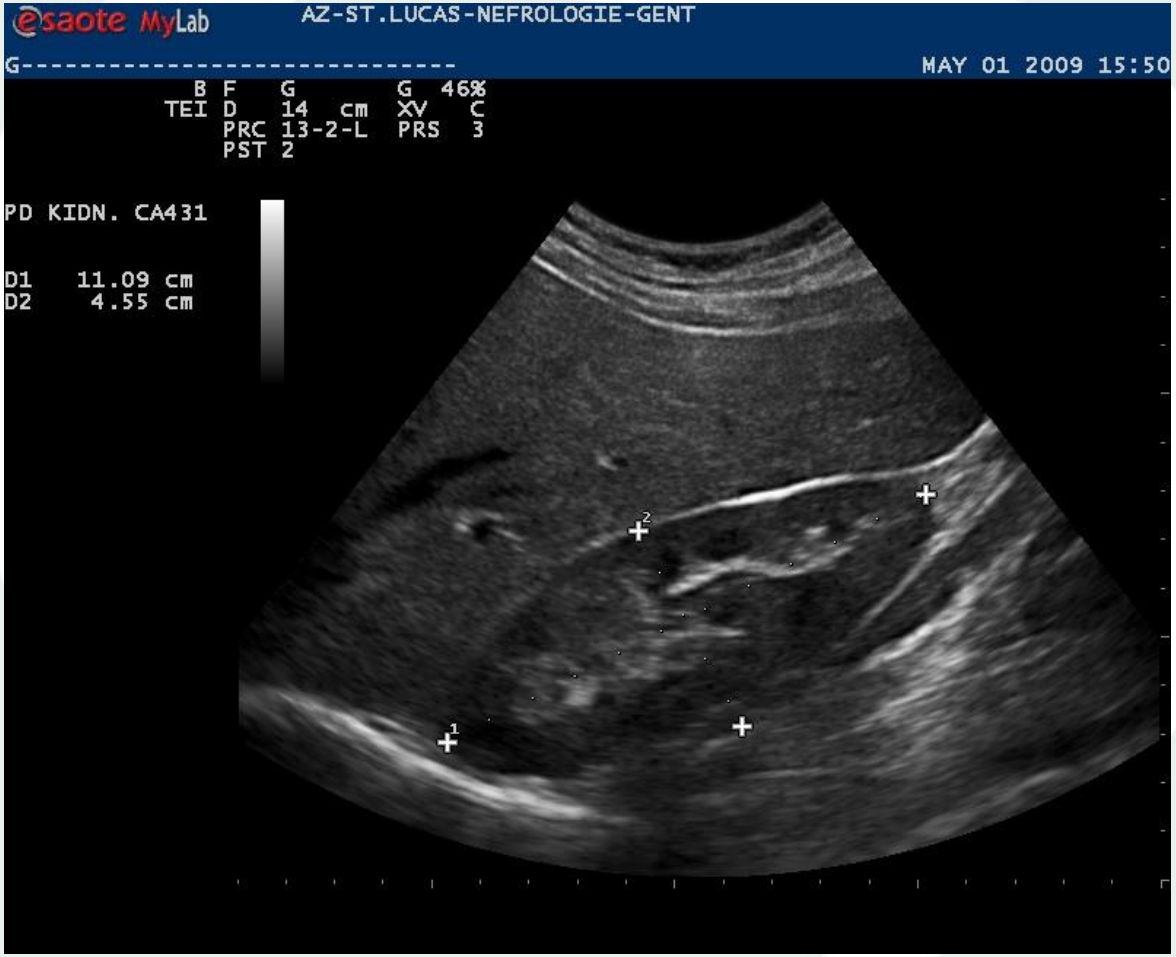
- <u>Rule of thumb:</u>

  Volume (ml) =

  Body weight (kg) x 2 +- 20%

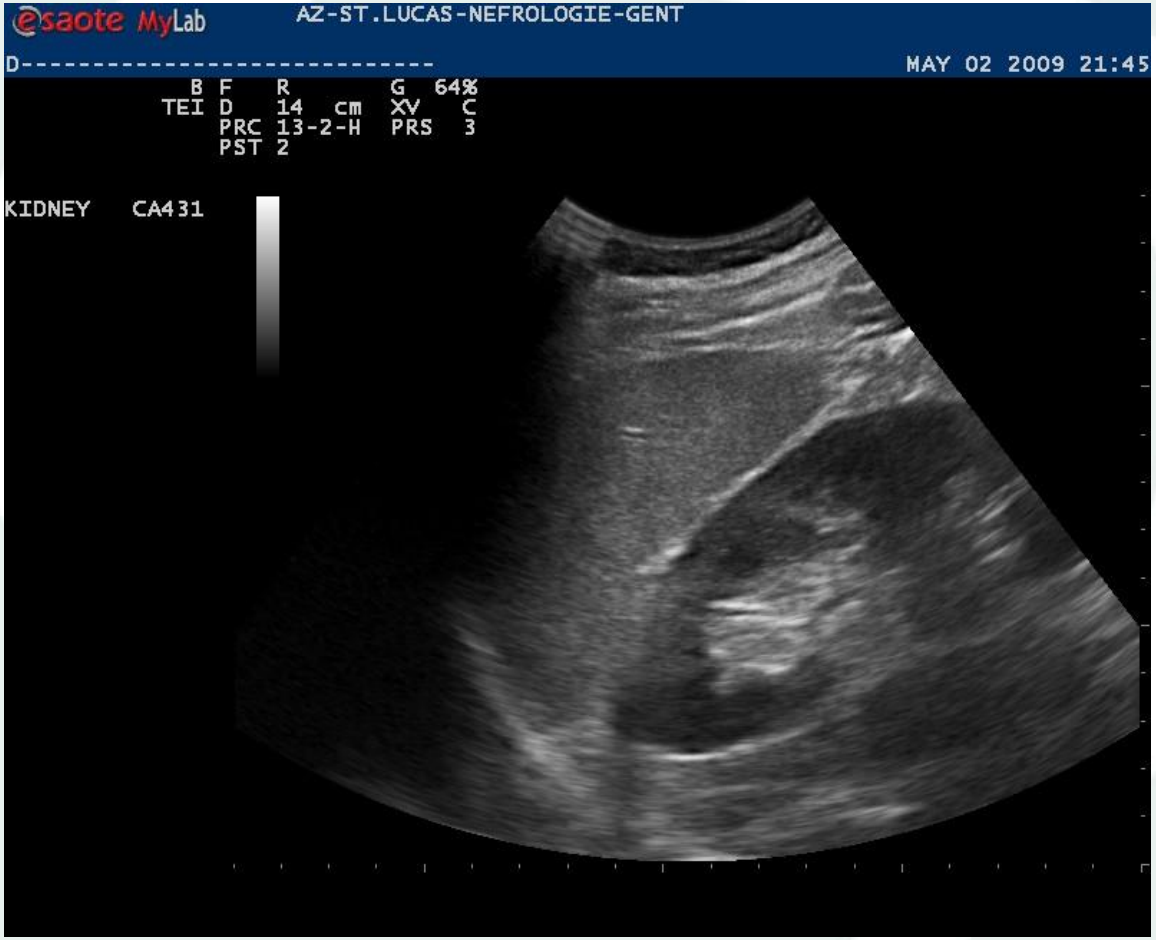
- Fex Male 75 kg -> kidney volume : 150 ml (120-180 ml)

Echogenicity R kidney



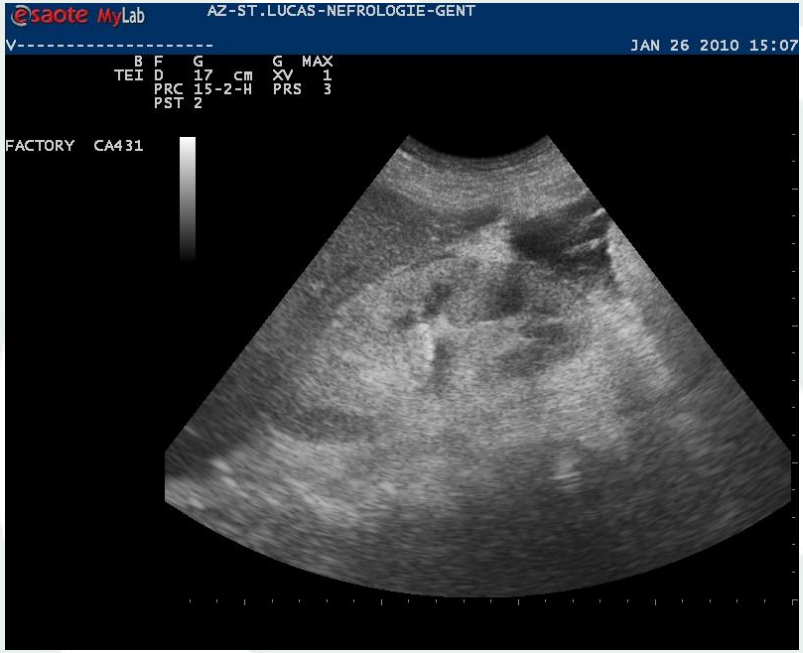
Normally iso- to slightly hypoechogenic compared to the liver.

Echogenicity left kidney

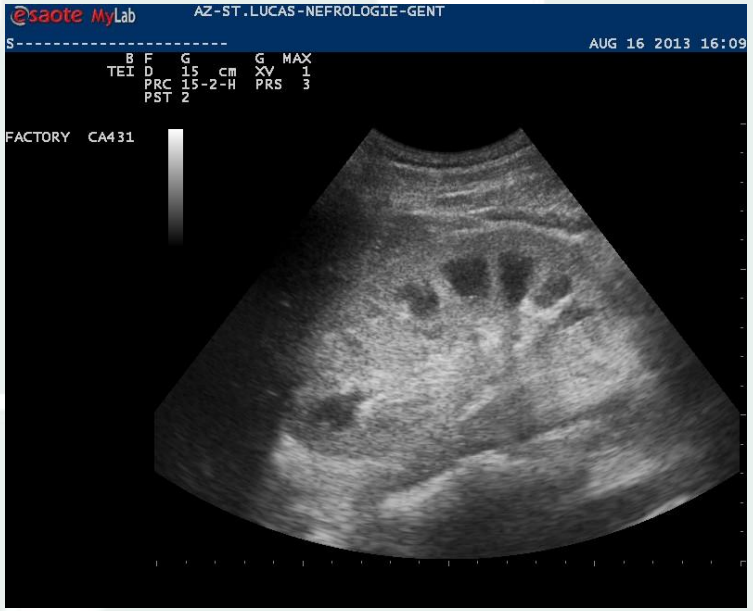


Normally iso- to slightly hypoechogenic compared to the spleen.

Hyperechogenic right kidney



=sensitive but unspecific sign of renal disease.

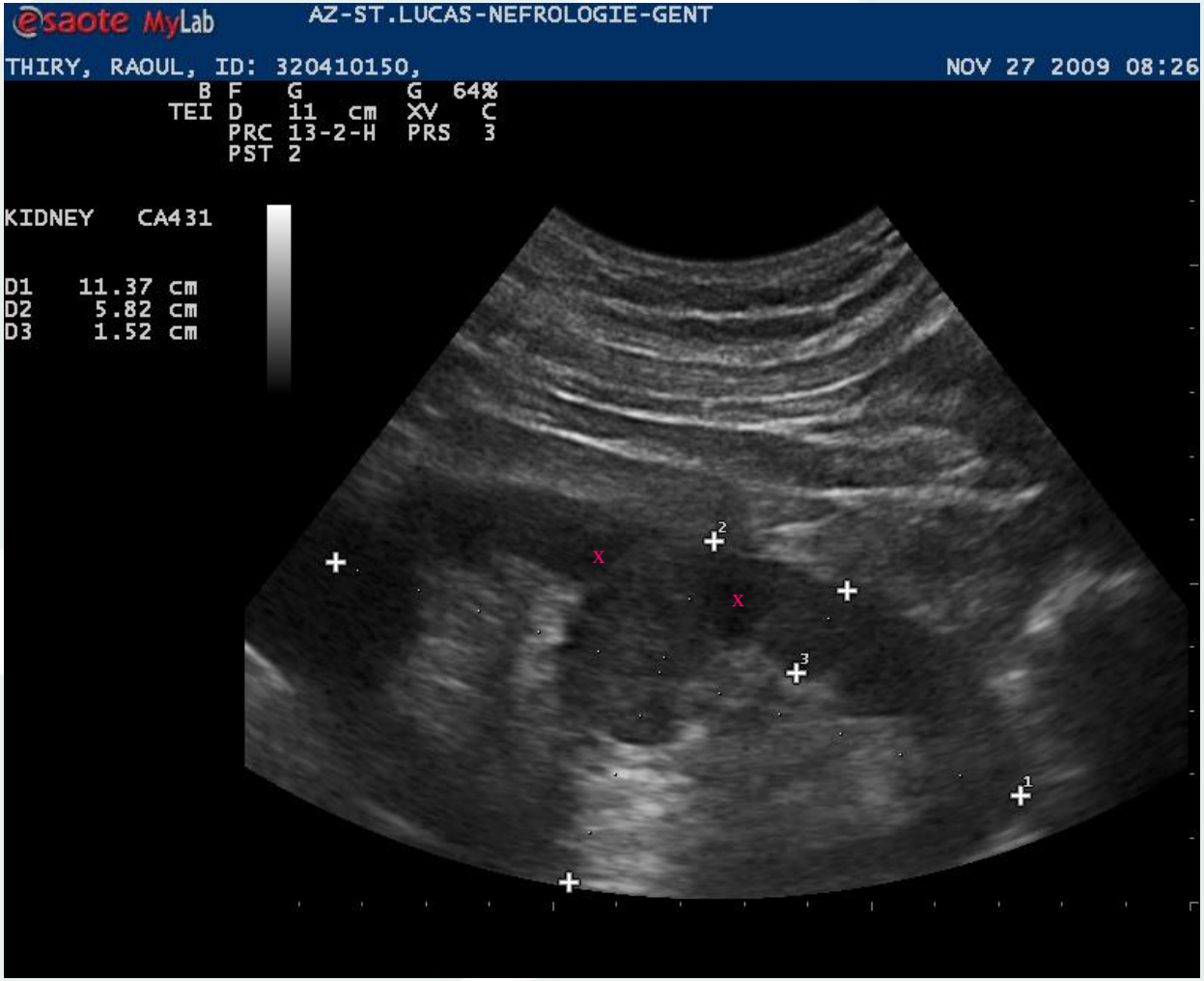


Caveat: upto the age of 6 months, renal parenchyma is hyperechogenic compared to liver.

(Normal) anatomical variants

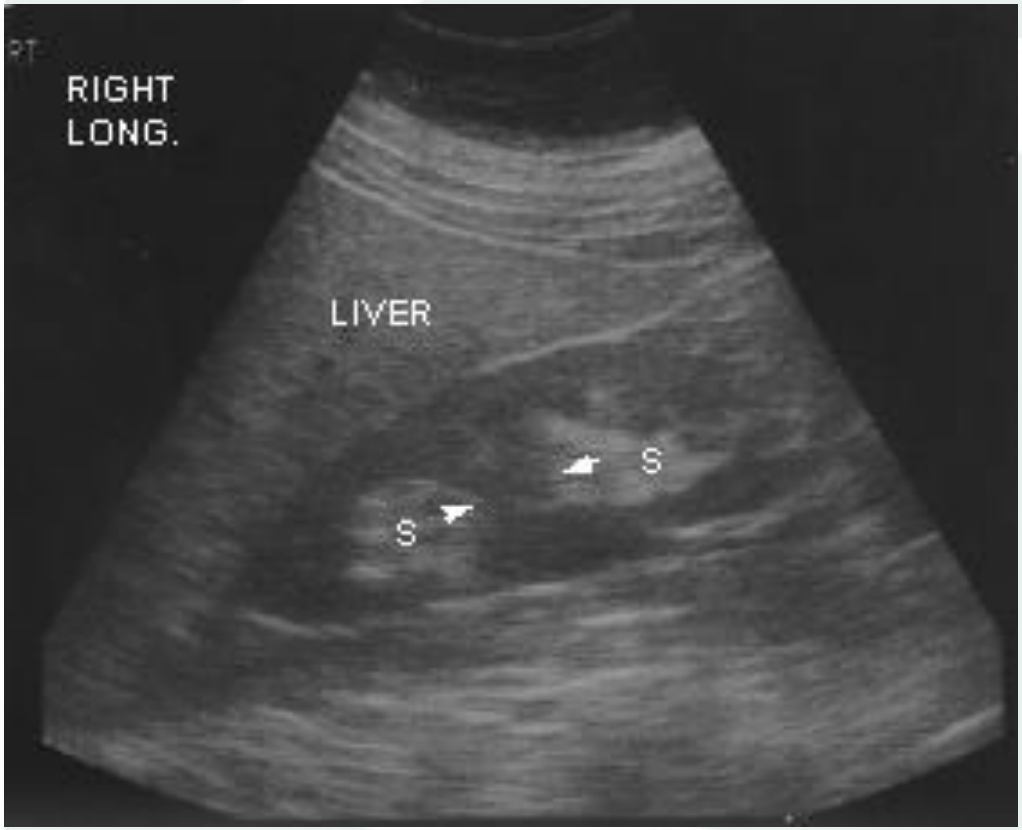
- Congenital a/ hypo genesis
- Hypertrophied column of Bertin
- Renal duplication (partial - complete)
- Dromedary hump
- Junctional fusion defect
- Horse shoe kidney
- Persistent fetal lobulation
- Ectopic kidney

Hypertrophied column of Bertin



X: medulla

Duplicated collecting system

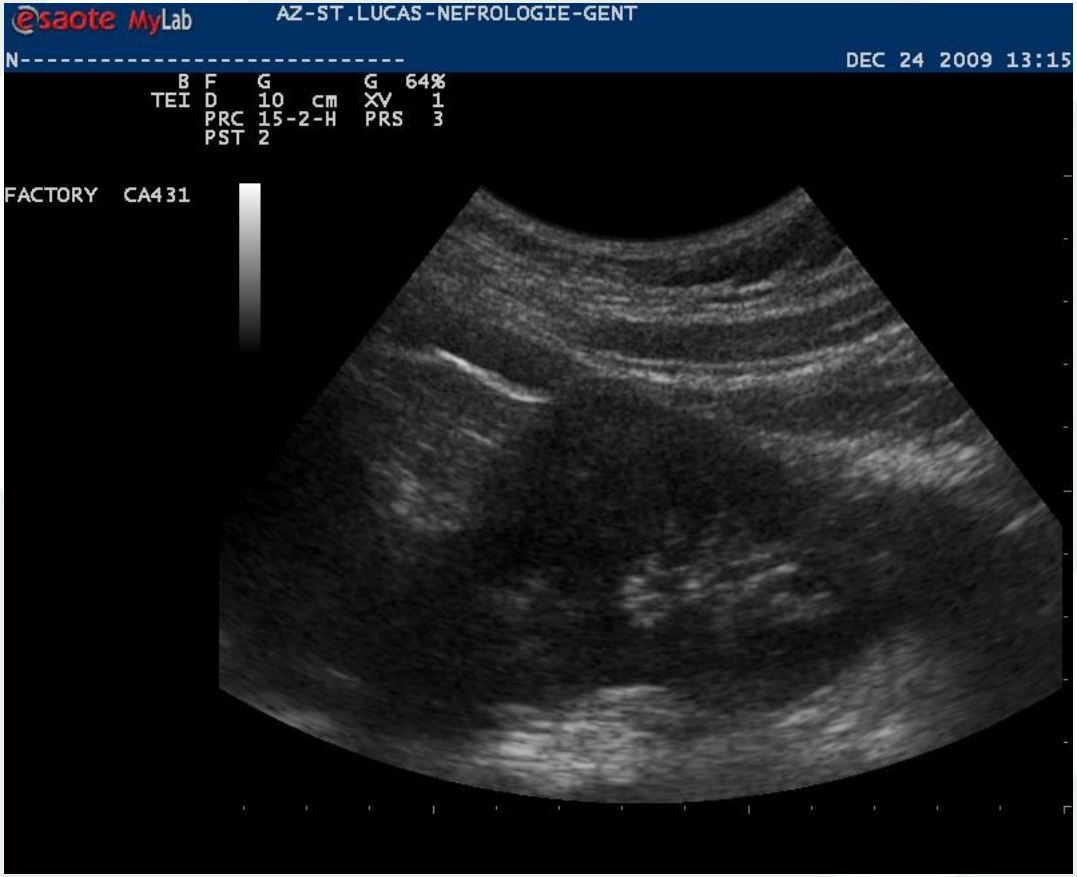


- Most frequent congenital malformation (0,5-10%)

- Cave : Reflux/Obstruction

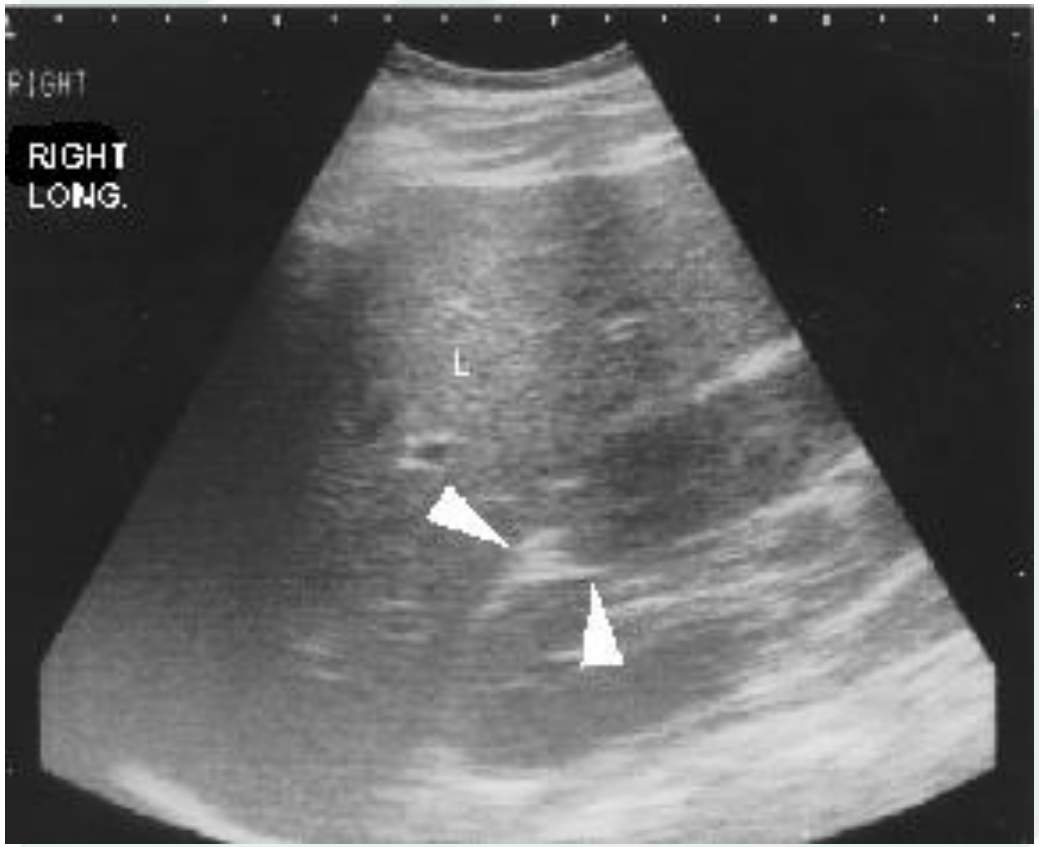
R kidney. There is a parenchymal bridge of cortex tissue (arrows) passing through the sinus, creating 2 separate sinuses (S). The kidney otherwise looks normal. Atlas of renal Ultrasonography (O'Neill)

Dromedary hump (splenic notch)



-Left kidney
-Wide parenchyma in middle portion
-DD renal mass

Junctional fusion defect

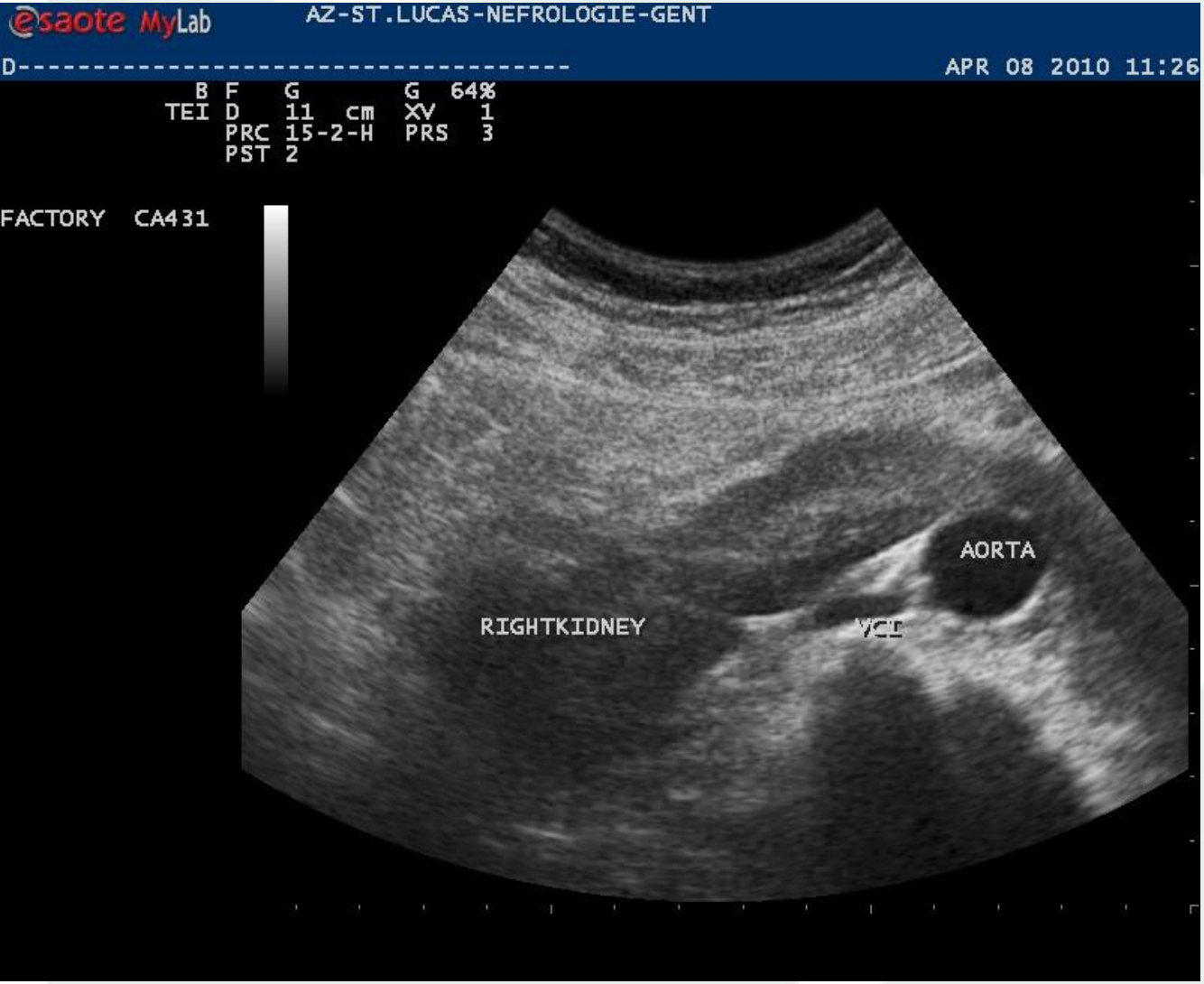


- R kidney,upper pole
- Anterior
- Triangular, wedge shaped
- Hyperechogenic
- In continuity with sinus

- DD Scar, mass

The junctional fusion defect (arrowheads) is a wedge-shaped defect in the cortex that is filled with echogenic fat in continuity with the sinus fat (lower arrowhead).
Caused by partial fusion of renunculi (embryonic parenchymatous masses)
Atlas of renal ultrasonography (O'Neill)

Horse shoe kidney

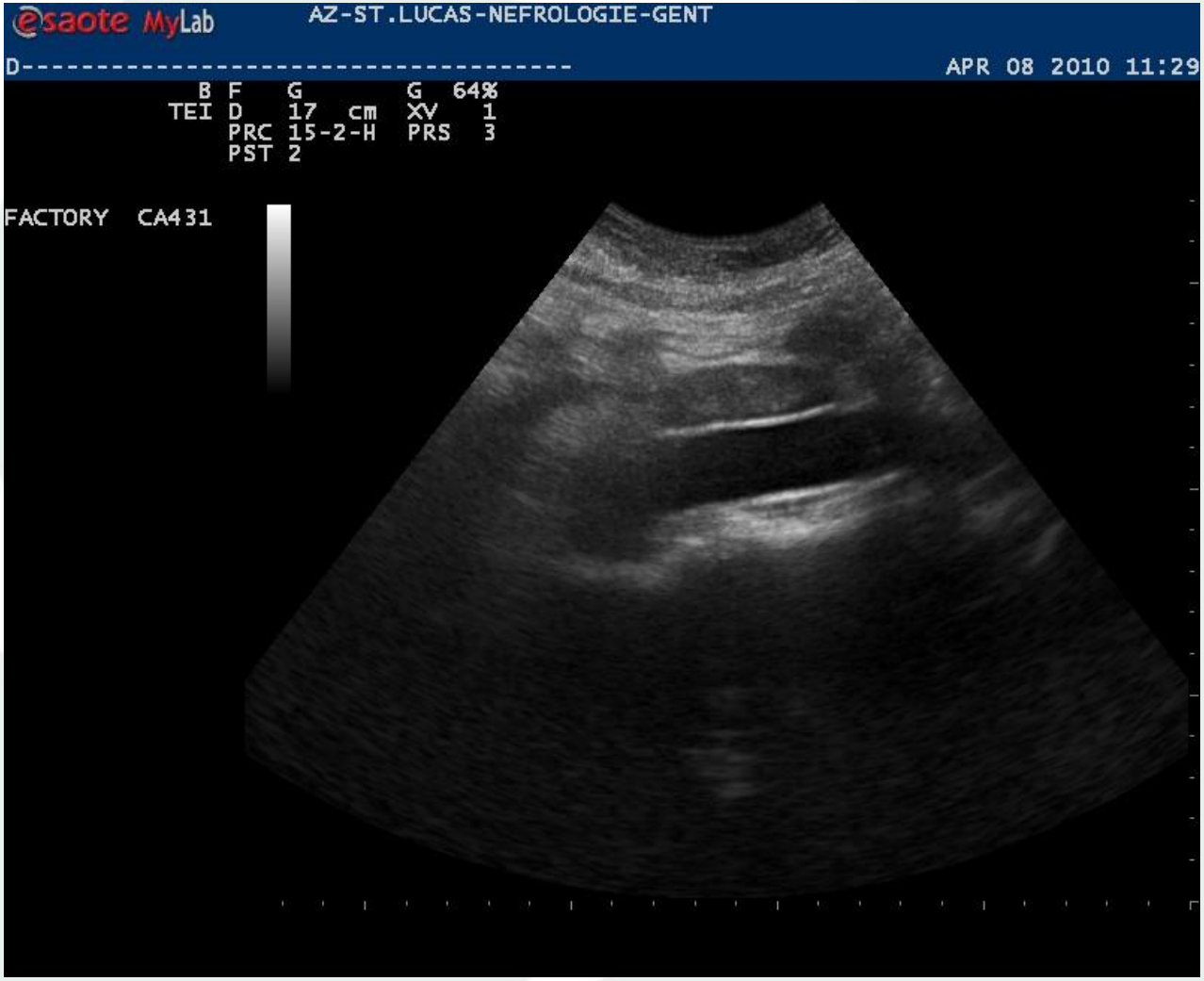


Fused lower poles

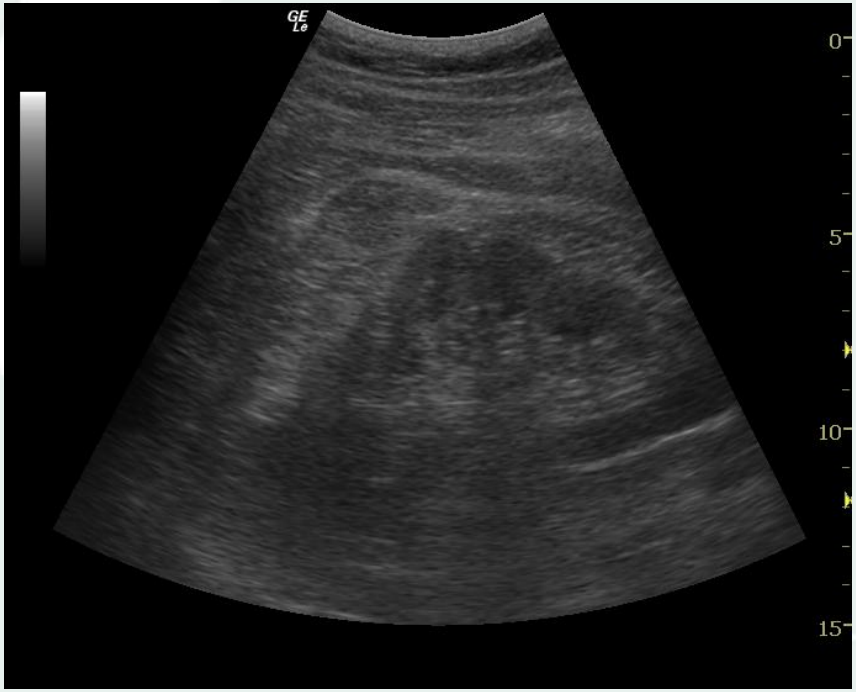
Cave :

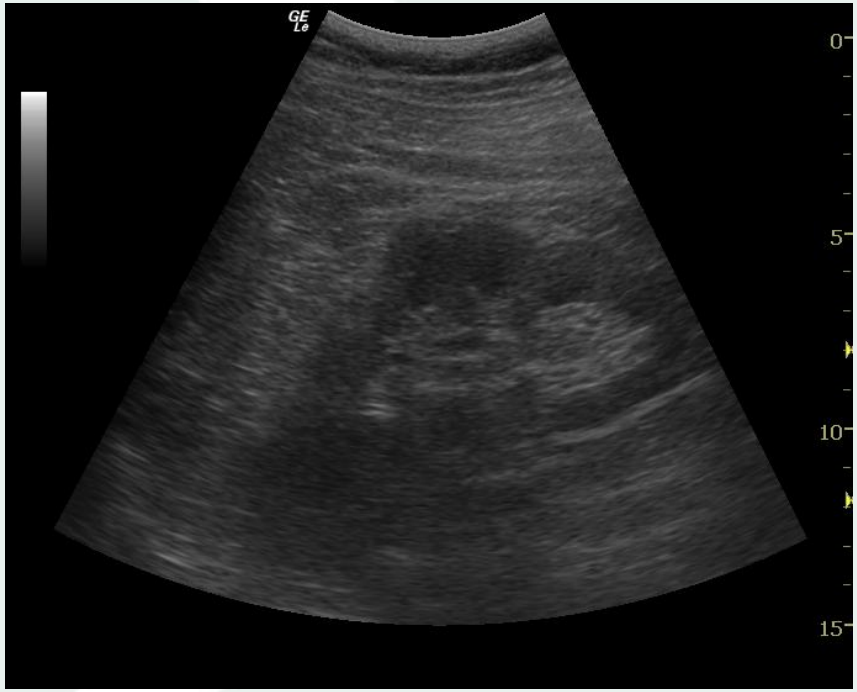
-VUReflux
-lithiasis
-urinary tract obstruction

Horse shoe kidney



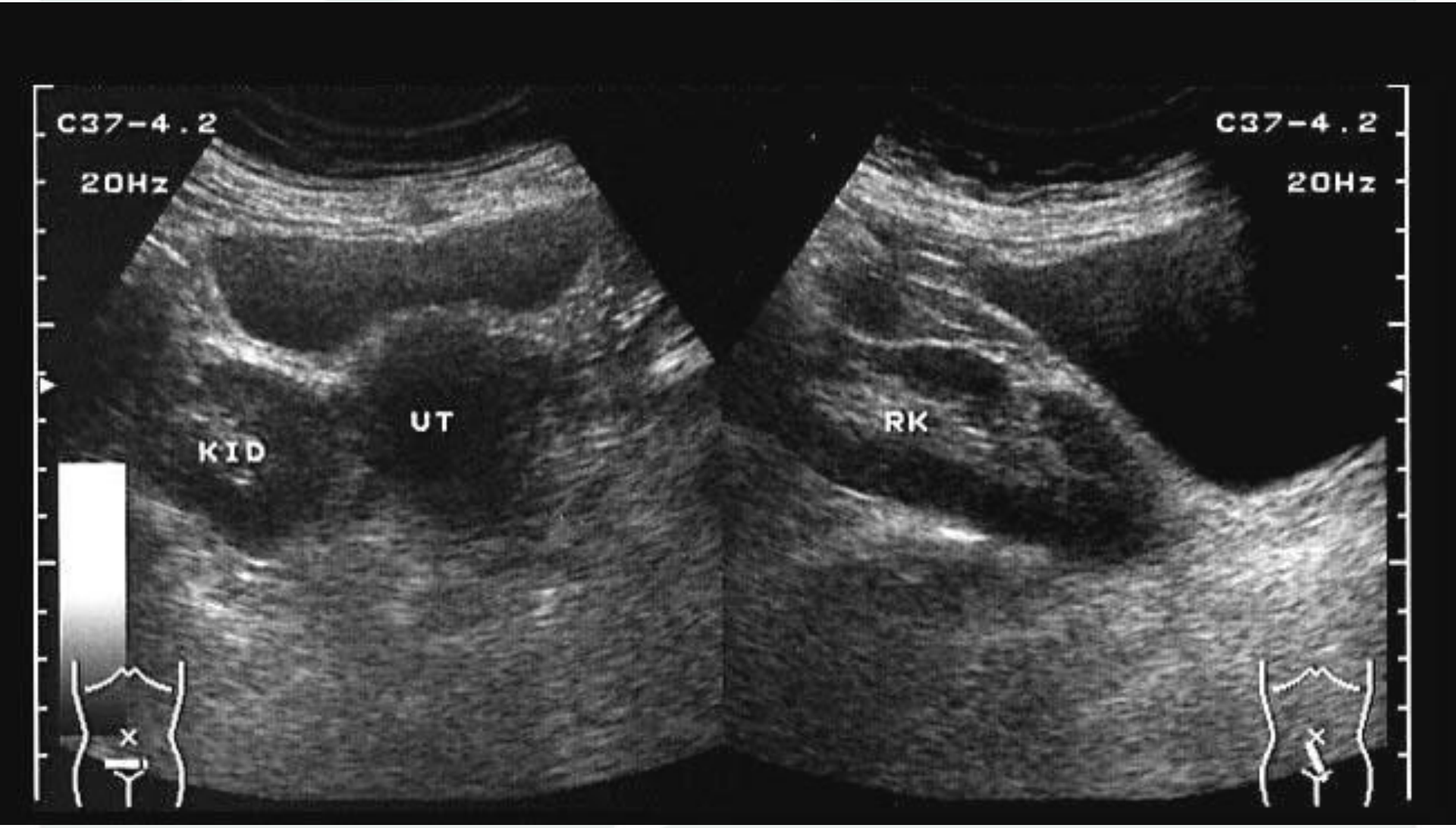
Persistent fetal lobulation





- Remaining signs of fetal lobulation
- More easy recognisable in the R kidney

Ectopic (pelvic) kidney



Renal B Mode US: pay attention to

- Size / volume
- Echogenicity
- Kidney shape / kidney surface
- Kidney location
- Signs of obstruction?
- Focal lesions?: stones/tumours/cysts

The 2 most important points of interest when looking a kidney

Renal echopathology

- Renoparenchymatous disease

- Focal lesions:
  - lithiasis (hydronephrosis)
  - cysts
  - tumours

Renoparenchymatous disease

- Unilateral vs bilateral disease
- Small vs enlarged kidneys
- Some general rules of thumb:

->small kidneys = chronic kidney disease

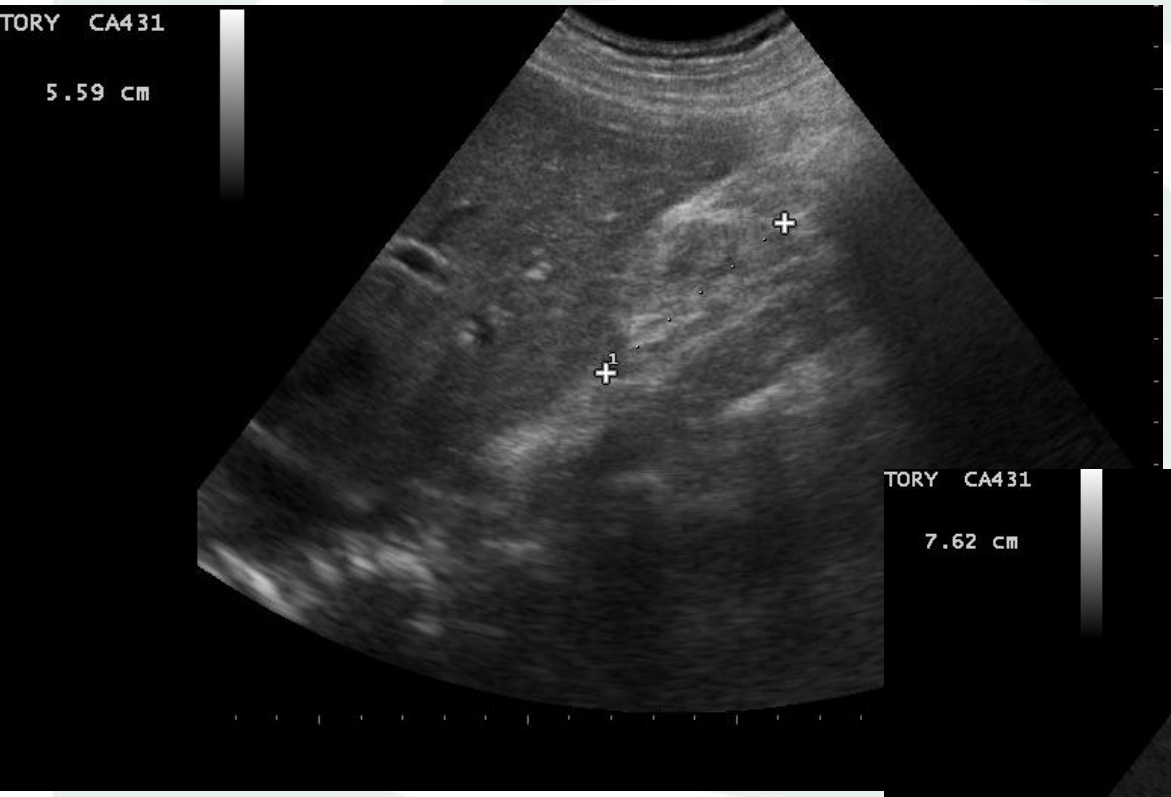
->enlarged kidneys = acute (potentially reversible) kidney disease

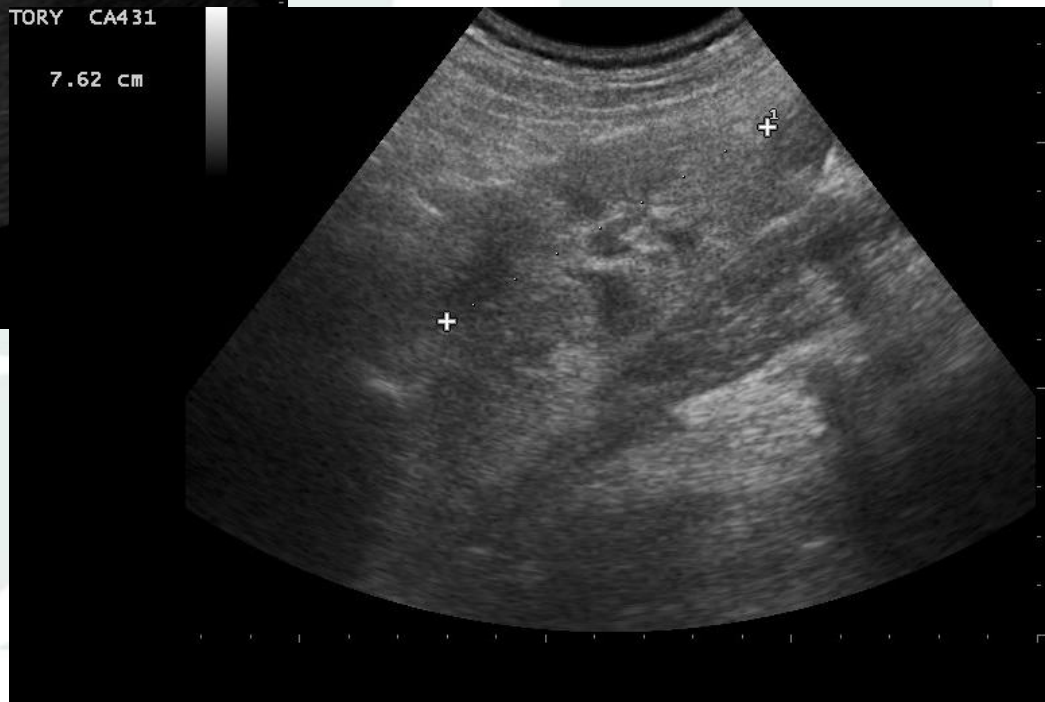
-> diseased kidneys = often hyperechogenic aspect of parenchyma

Case 1: F, 50 yo, Guadeloupe

- Nocturnal presentation on ED
- Dyspnea, vertigo, tired, nausea, cramps
- BP 210/160 mmhg ; lungs: fine creps bibasally
- History: non treated AHT since 10 years
- Lab: s creat 12 mg% Ureum 256 mg/dl
- Chest X-ray: cardiomegaly + congested hili
- ECG: LVHT

Renal US





Bilateral small kidneys: DD

* Terminal stage of almost all renal diseases

* Chronic GNF

* Chronic PNF

* Analgetic nephropathy

* Bilateral RAS

* Nefrangiosclerosis (AHT)

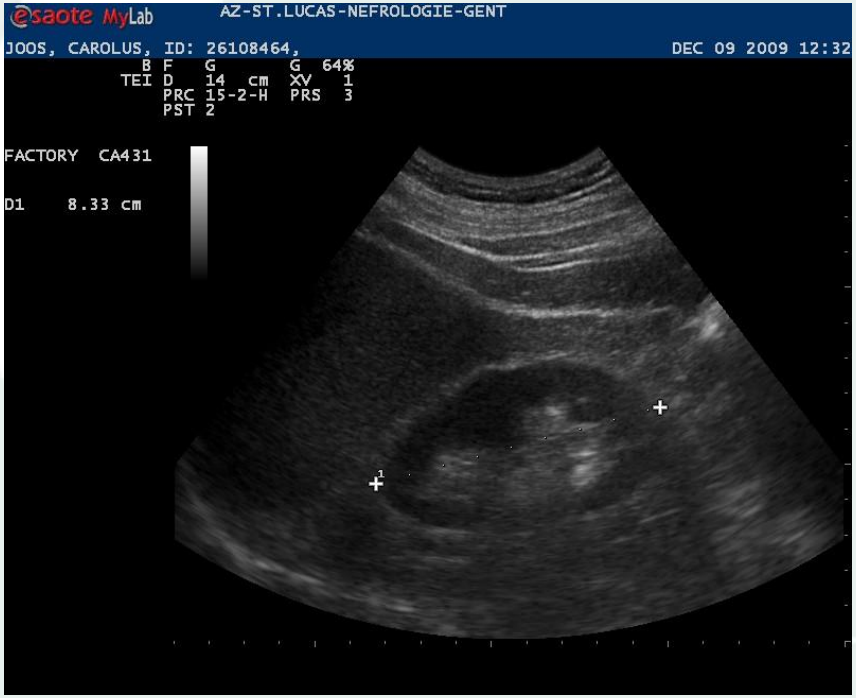
Case 2 : 53 yo male

- Mixed dyslipidemia
- Heavy smoker: 40 py
- 10-2008: stent R common iliac artery
- 08-2009: idiopathic dilated CMP (EF 20%)
- AHT since 1980, since 08-2009 poorly controlled despite 4 antihypertensive R/
- A on CRF: s creat 1.33 mg% 08-2009

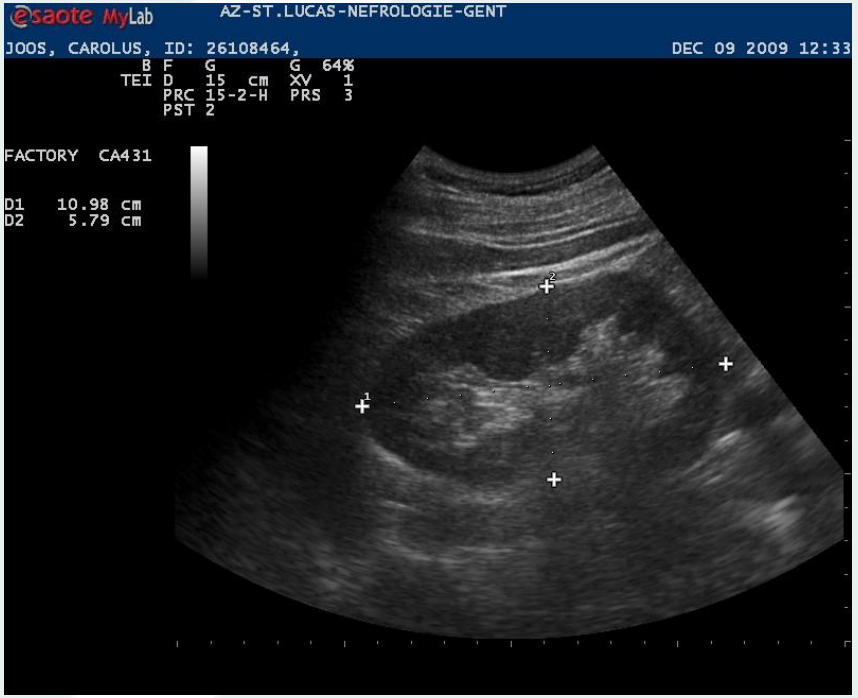
  s creat 2.74 mg% 12-2009

B mode renal US

R kidney

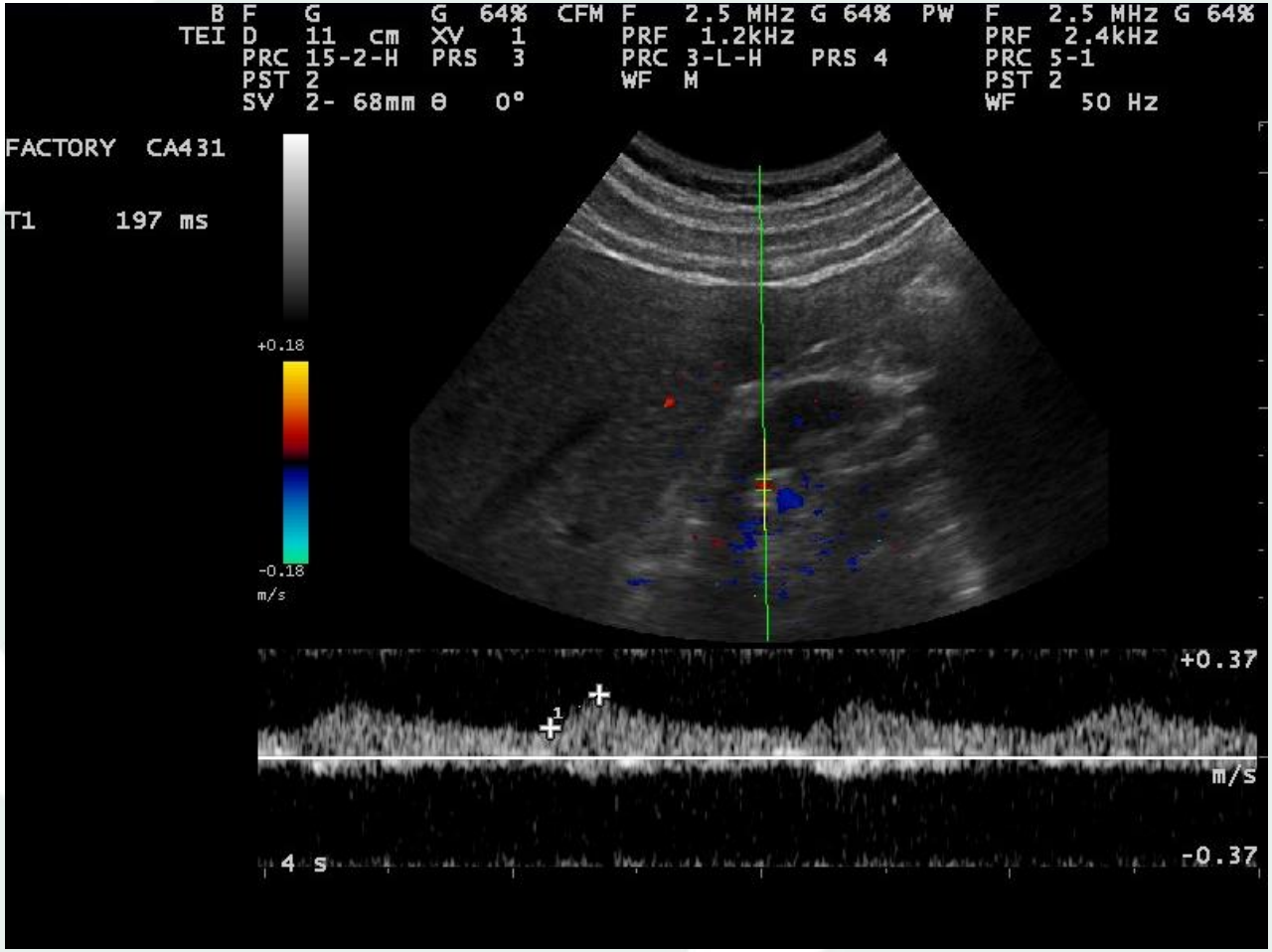


Left kidney



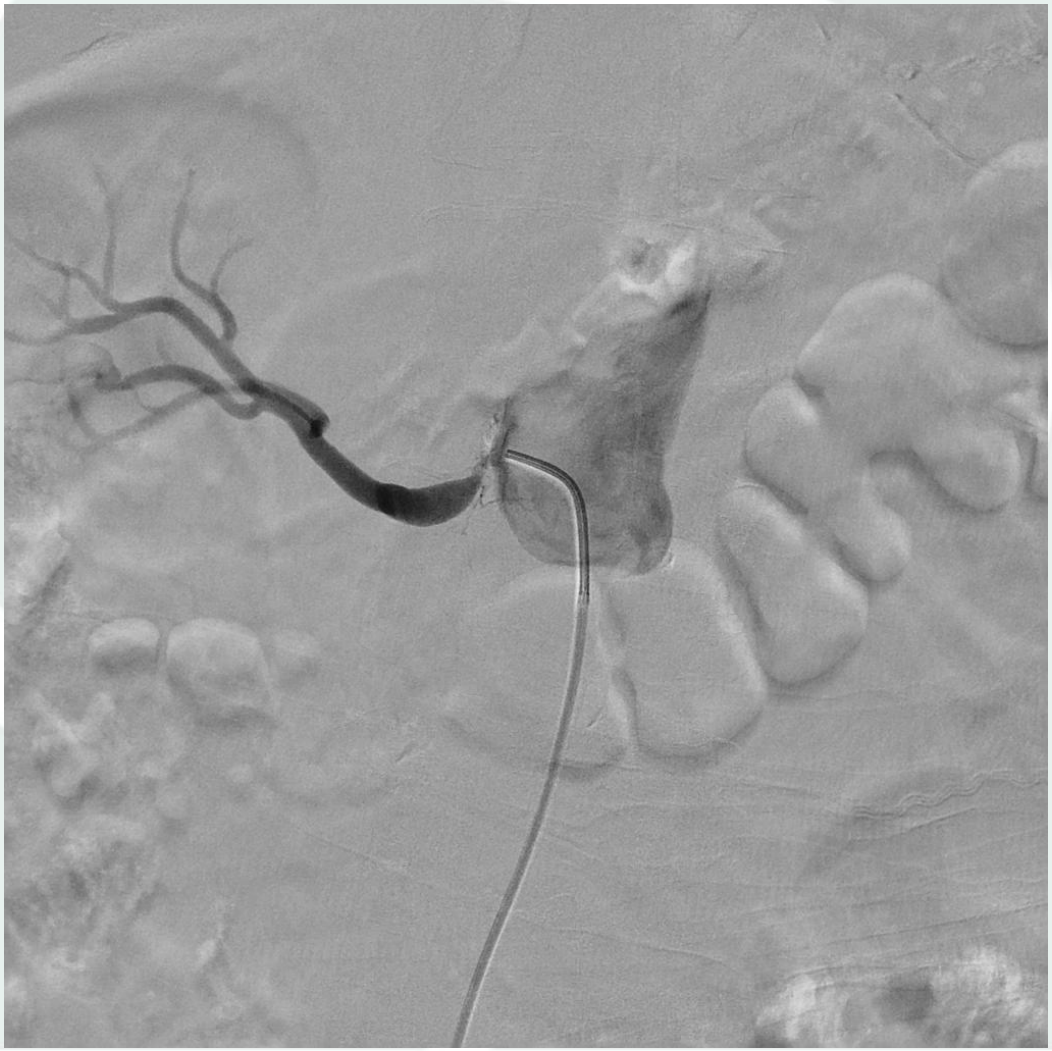
Clearly asymmetric !

Duplex R kidney



Tardus parvus wave form R intrarenally
AT 197 msec en RI: 0.44

95% stenosis R renal artery

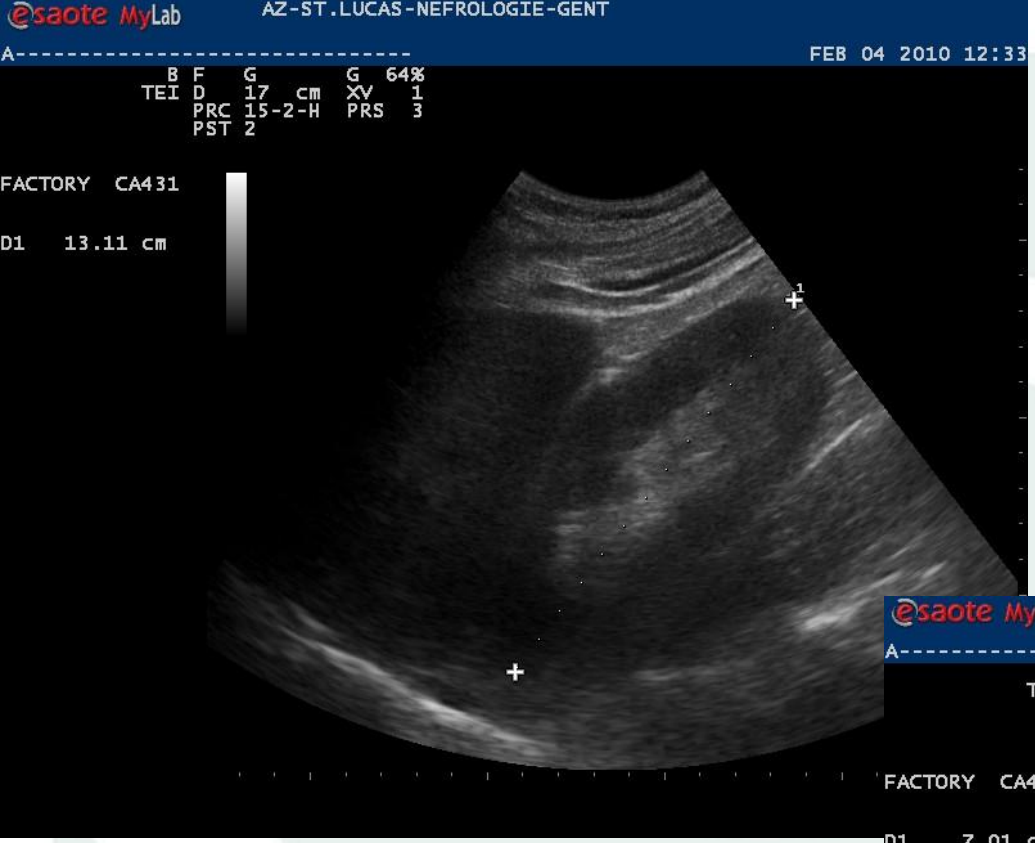


Unilateral small kidney: DD

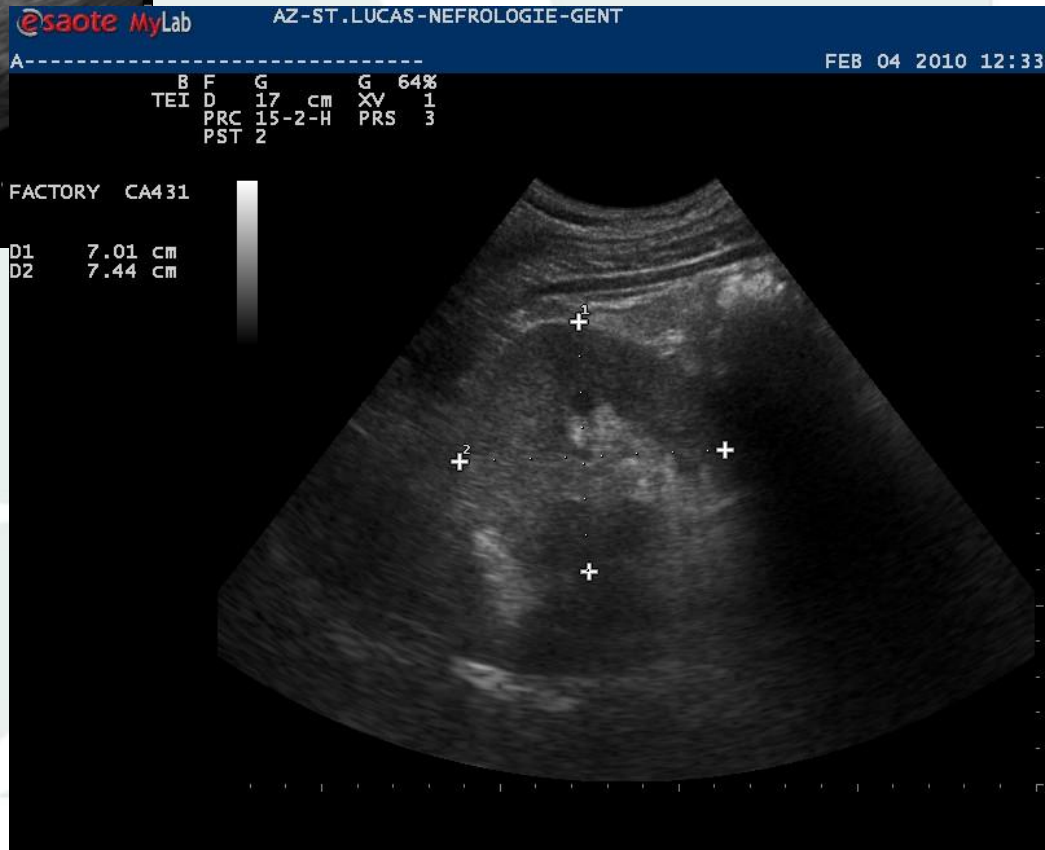
- Hypoplasia – agenesis
- Renal irradiation
- Chron. Pyelonefritis
- Renal artery stenosis
- TBC

Case 3: Male, 36 y

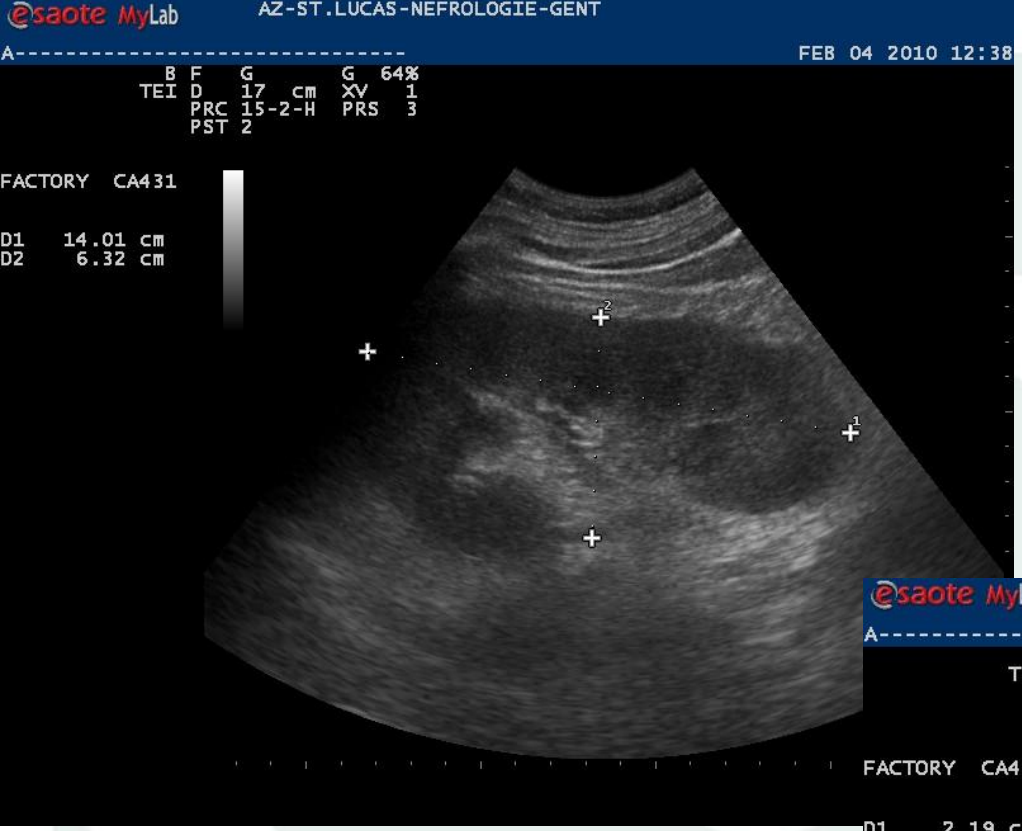
- Since 2 months diffuse edema (ankles; hands, face). More tired.
- BP 170/100 mmhg + pitting edema LL
- S creat : 1,01 mg% eGFR > 60 ml/min; Alb 1,8 g/dl, TG 398 mg/dl , Tchol 388 mg/dl
- Urine: EW/creat 8,8. RBC 19/field WBC 19/field



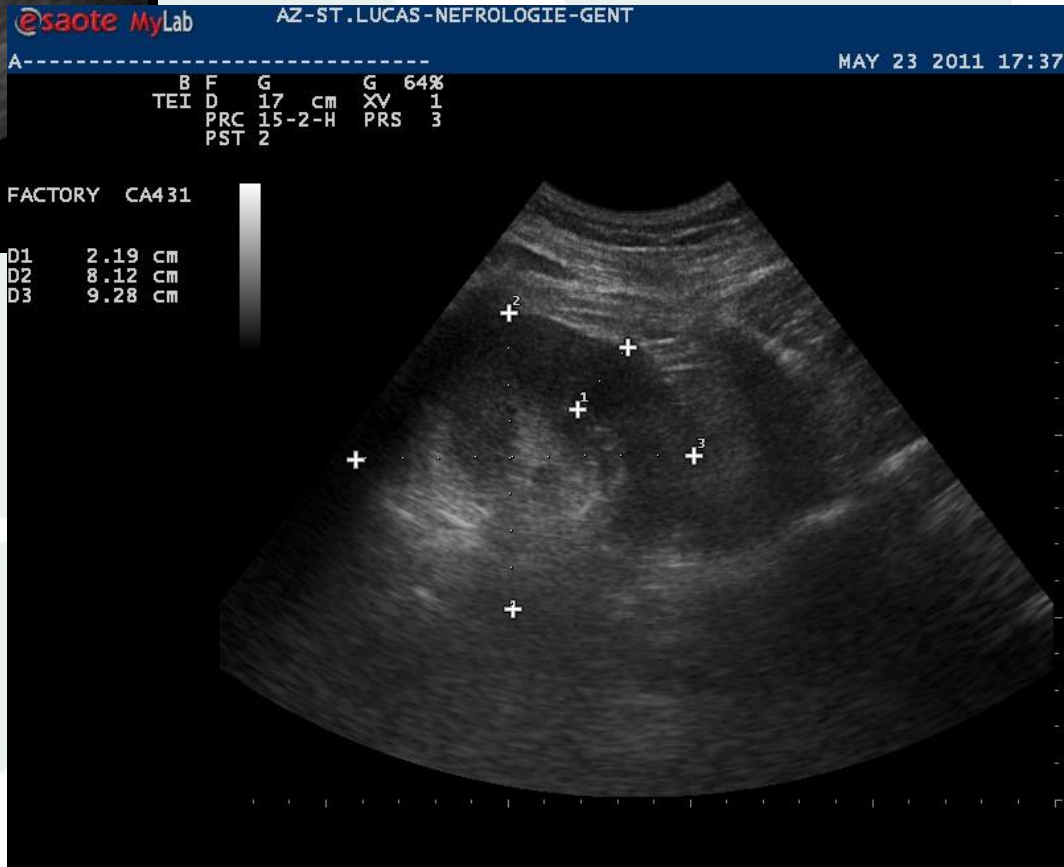
Weight = 80 kg
Normal kidney volume =
160 cc +- 20%
(128 – 192 cc)



Volume R kidney =
13 x 7 x 7.4 /2 =
336 cc



Volume L kidney =
14 x 8 x 9 / 2 =
504 cc



DD Glomerulonephritis/vasculitis

- No definite diagnosis with US
- Only with kidney biopsy
- DD can be narrowed combining the history, clin ex, lab and US findings

Bilateral enlarged kidneys: DD

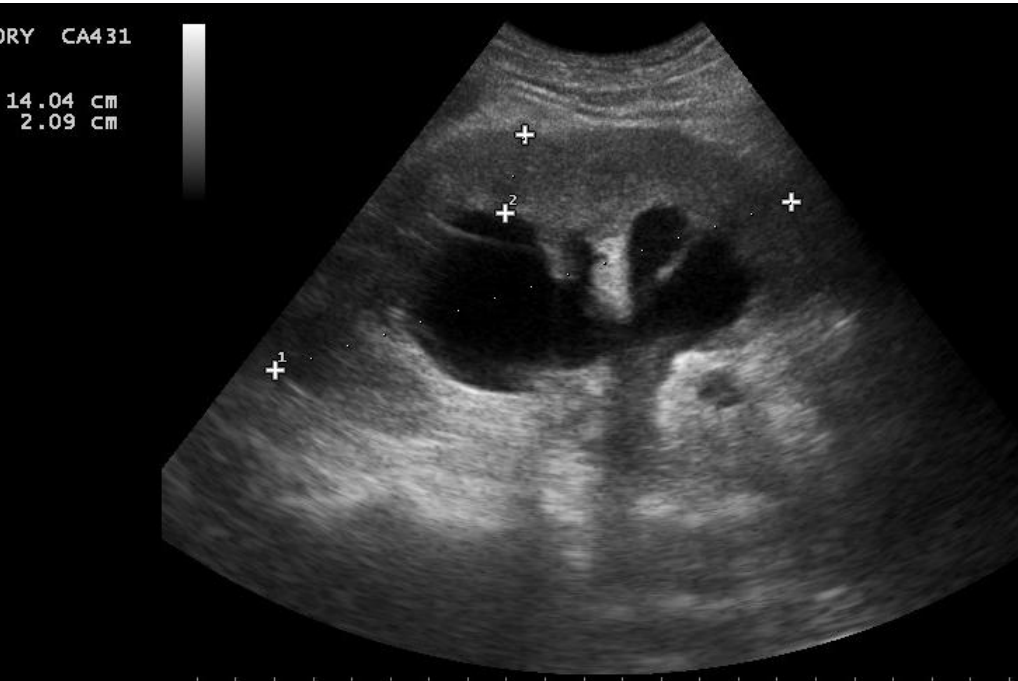
- All acute renal diseases
- Postvesical UT-obstruction
- Diabetes
- Amyloidosis
- Pregnancy
- Polycystic kidney disease

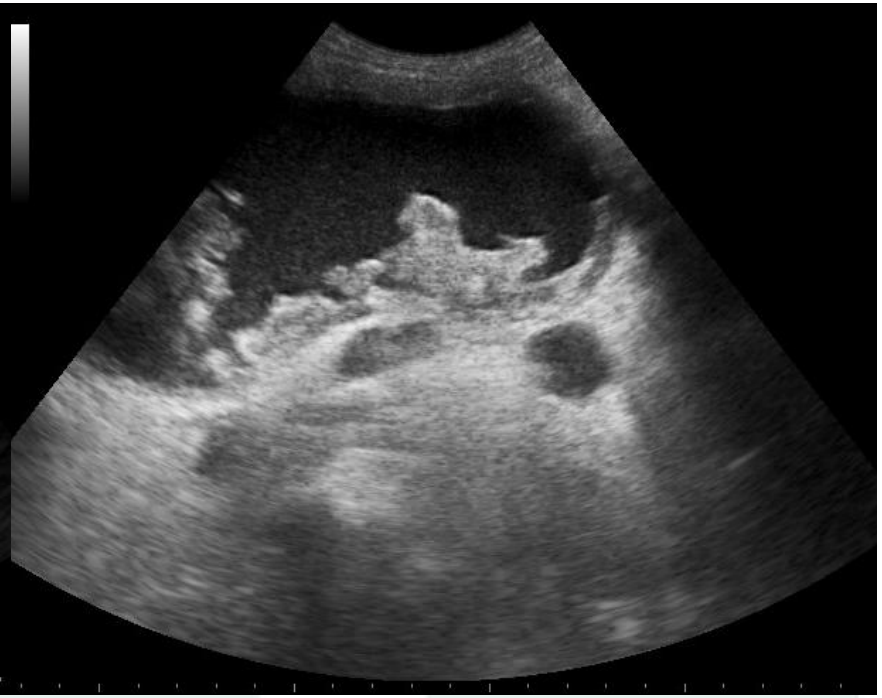
Diabetic nephropathy

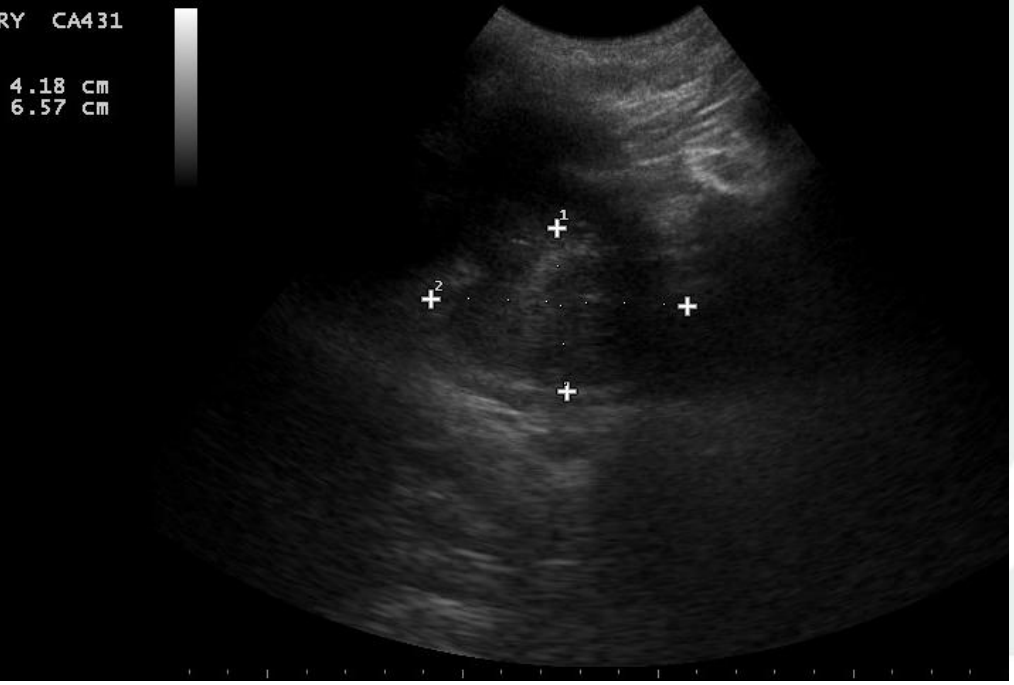
- Very frequent cause of ESRD (40%)
- Almost always associated with large kidneys in the pre ESRD stadium (DD with hypertensive nephrosclerosis)
- The worse the renal function, the more hyperechogenic is the renal parenchyma
- Even in ESRD, the kidneys can still have a normal size.

Case 4 : Male 63 y

- Presentation ED
- Nausea, weakness, dysuria since months
- R/ NSAIDs (back pain)
- S creat 8,8 mg% (09-2010)
- Renal US : Unique R kidney
  Hydro-ureteronephrosis
  Enlarged prostate
  Irregular bladder wall







Urological consult :

-Cystoscopy : enlarged prostate, trabeculated bladder, not suspected for carcinoma
-TURP -> APO: BPH
-Partial recovery of renal function (s creat 2.5 mg%; 04.2011)

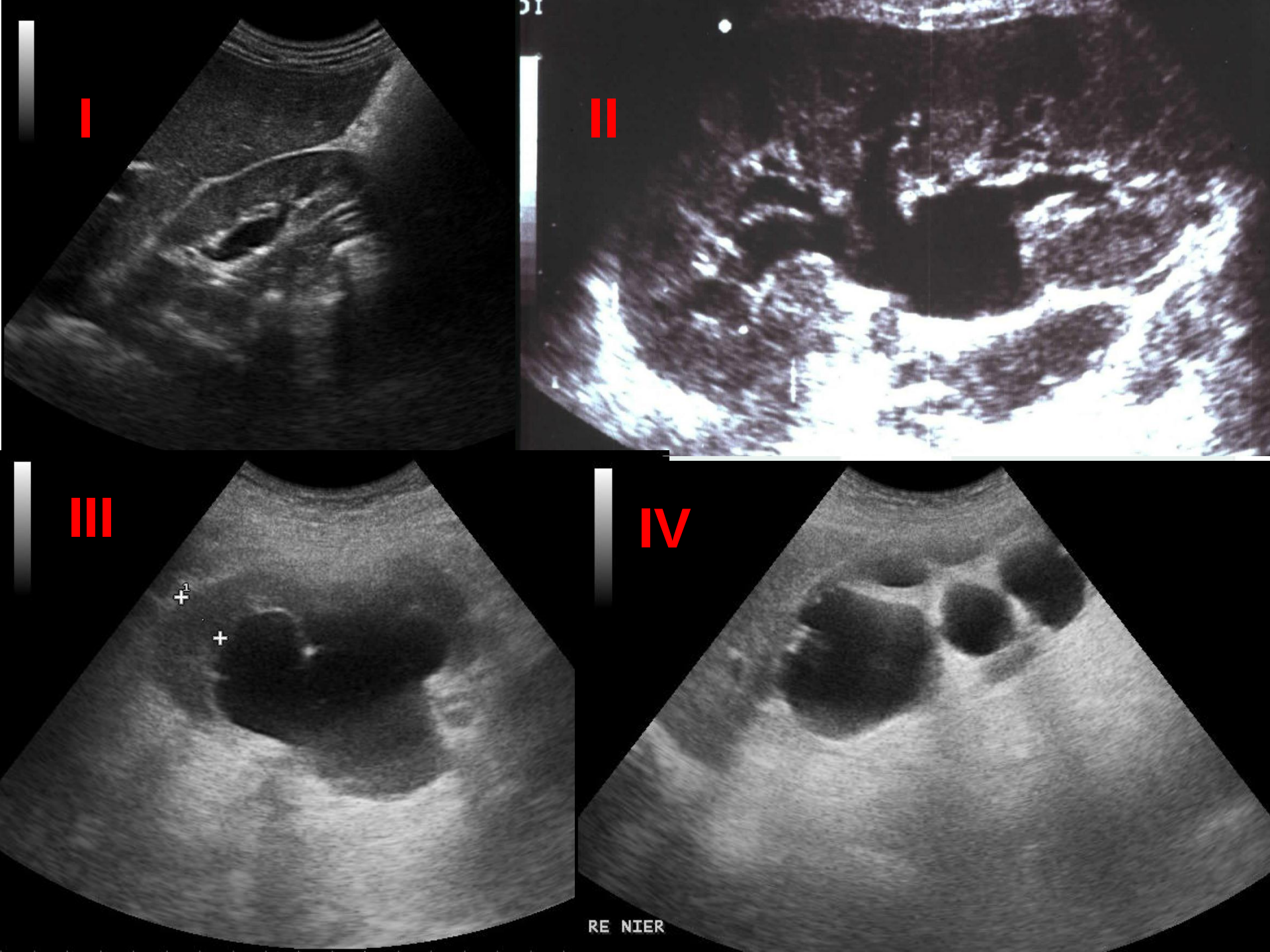
Unilateral enlarged kidney DD

- UT-obstruction
- Acute pyelonephritis
- Renal vein thrombosis
- Single kidney

Classification hydronephrosis

grade I	Only pyelectasia, no caliectasia, normal parenchyma
grade II	Pyelectasia, only a few calices are dilated, normal parenchyma
grade III	Pyelectasia, extensive caliectasia – normal parenchyma
grade IV	As grade III, thin parenchyma

Fernbach et al. 1993 Pediatr Radiol;23:478-80


I
II
III
IV
RE NIER

HYDRONEPHROSIS

- Unilateral or bilateral ?
- Do you see lithiasis?
- How do bladder + prostate look like?

->Globus vesicalis?

->Irregular bladderwall lining?

->Enlarged prostate?

- Repeat US after miction

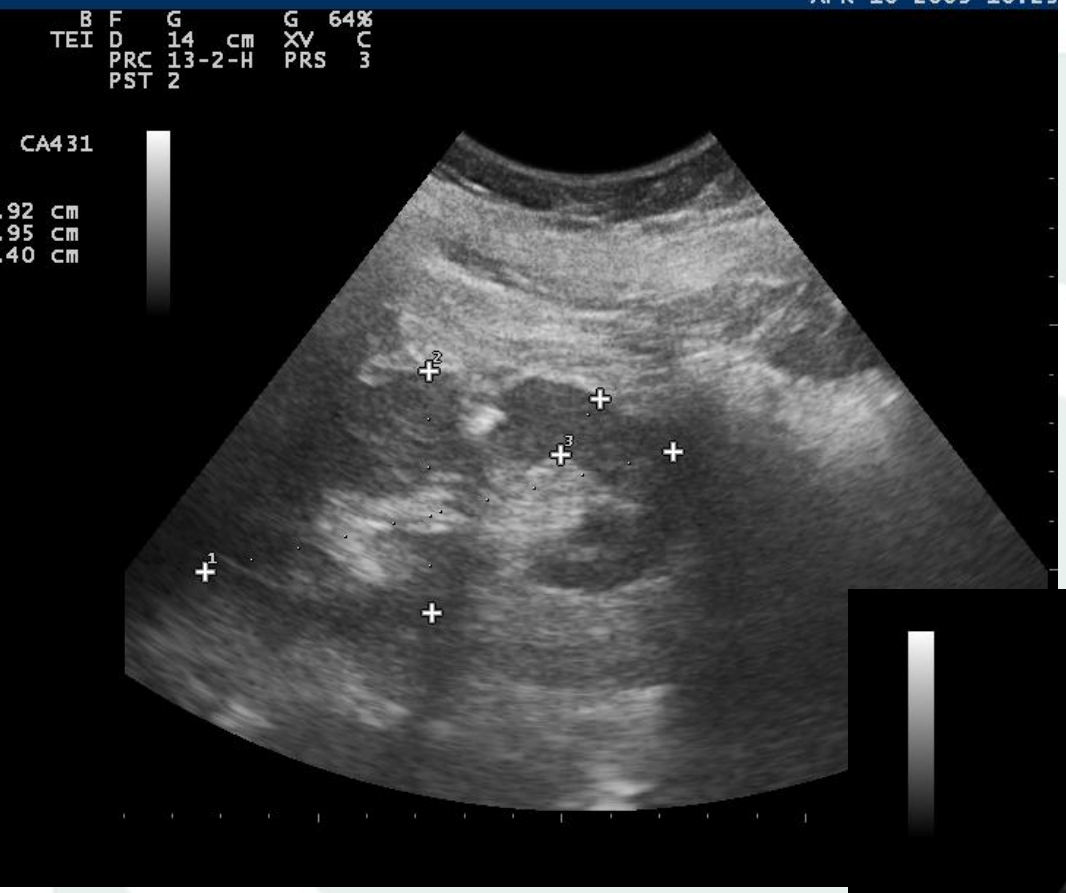
-> postmictional residu? Disappears hydronephrosis after miction?

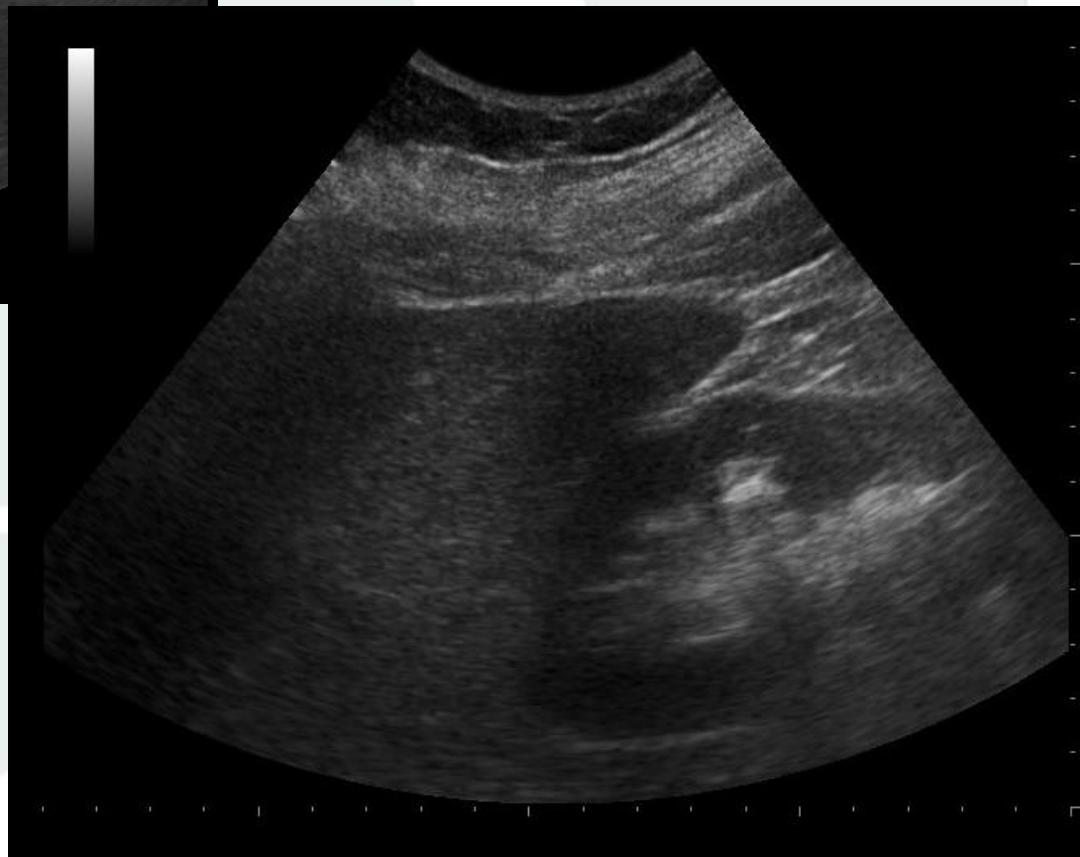
- (Measure the intrarenal art RI)
- Cave hydronephrosis w/o obstruction: pregnancy, diabetes insipidus, tx kidney

Reflexes

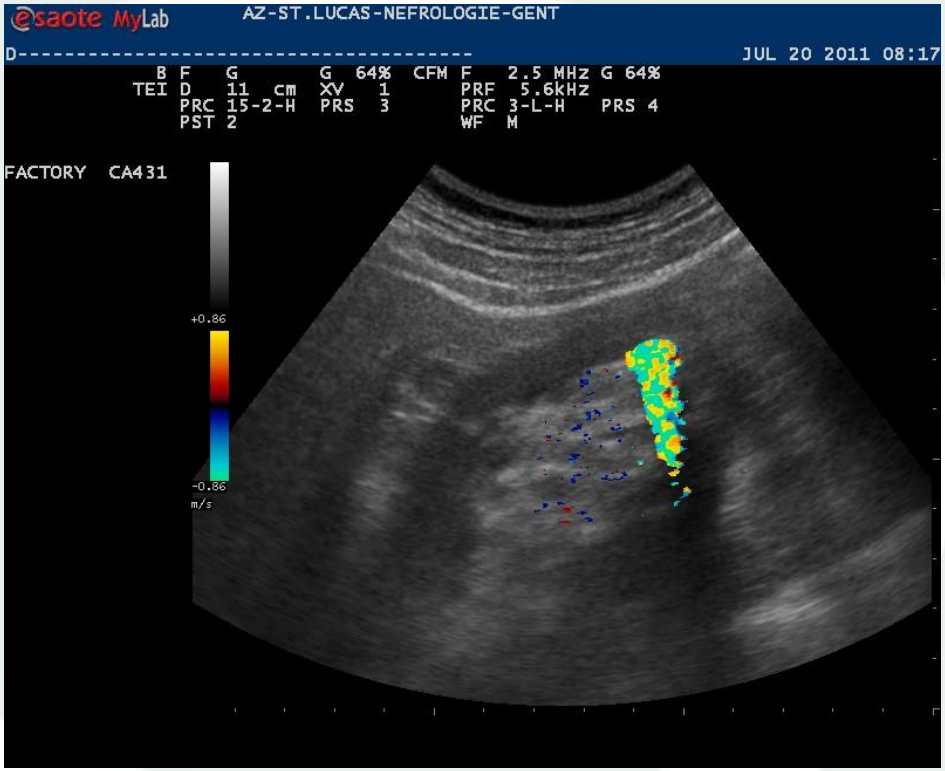
Lithiasis

- Similar ultrasonic appearance regardless of stone type.
- Size determines ease of detection.
- Stones > 5 mm can be reliably detected with US.
- More easy to find with a higher frequency probe.
- Are hyperechogenic with retro-acoustic shadow.
- Twinkle artefact.
- Are harder to find and evaluate than galbladder stones.
- CT à blanc = better examination than US to look for stones


B F G G 64%
TEI D 14 cm XV C
PRC 13-2-H PRS 3
PST 2
CA431
.92 cm
.95 cm
.40 cm



Twinkle or comet tail artefact



Tips to better visualize the comet tail artefact:

1. Set a low color doppler frequency
2. Increase the colour scale (elimination of the normal frequency shifts)

Cysts

- Most frequent observed renal laesion
- Most are trivial (« simple cyst »)
- Prevalence increases with age
  (20% at 50 y)
- Autopsy studies : up to 50% !
- 2x as frequent in males than in females

Ultrasound = less sensitive then CT to detect a renal mass

But the value of US =

To distinguish

-A benign cyst

From

-A more complex cyst / solid mass

Classification of renal cystic disease

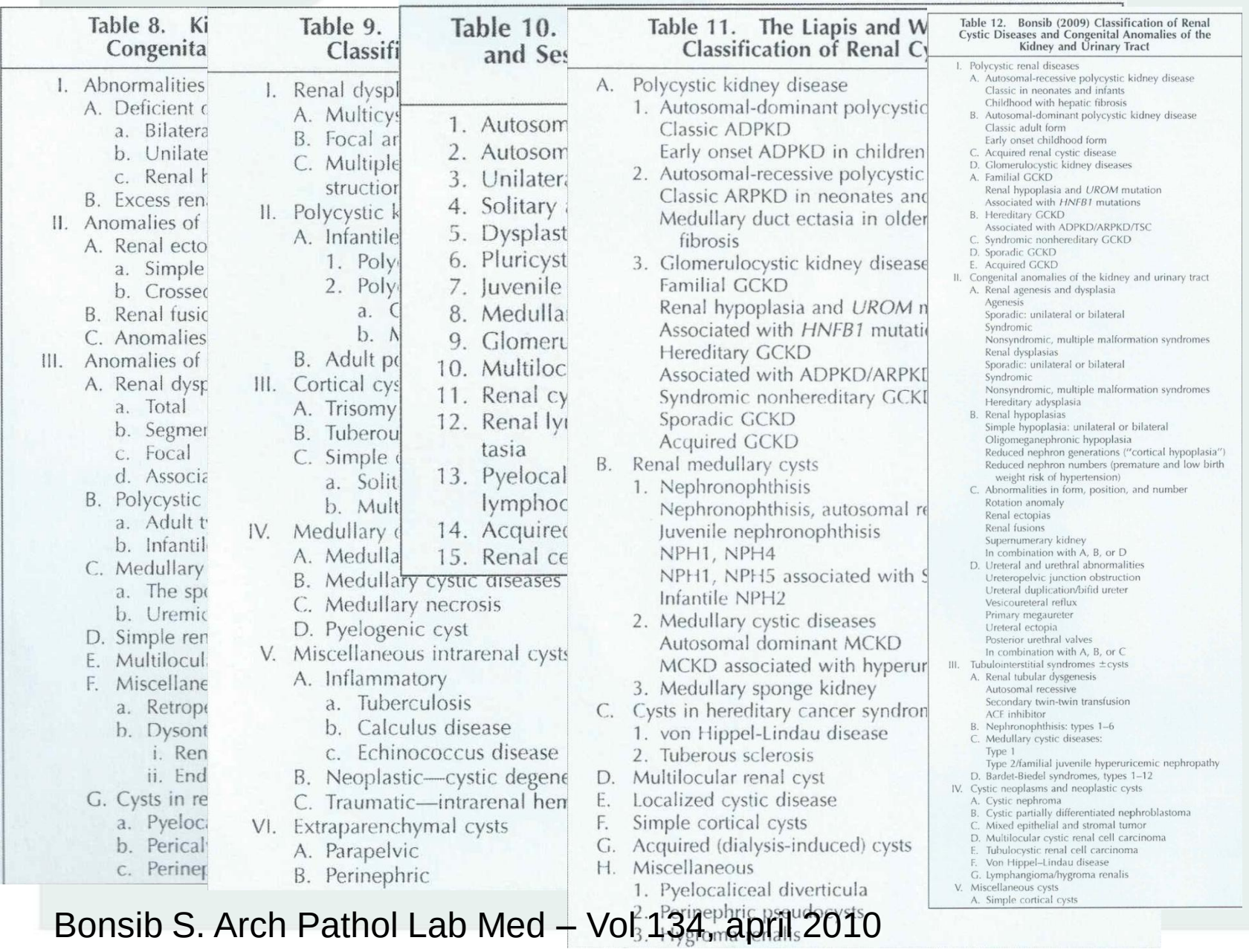


Table 8. Ki… Congenita

- I. Abnormalities
  - A. Deficient
    - a. Bilatera
    - b. Unilate
    - c. Renal
  - B. Excess ren
- II. Anomalies of
  - A. Renal ecto
    - a. Simple
    - b. Crosse
  - B. Renal fusic
  - C. Anomalies
- III. Anomalies of
  - A. Renal dysp
    - a. Total
    - b. Segmer
    - c. Focal
    - d. Associa
  - B. Polycystic
    - a. Adult t
    - b. Infantil
  - C. Medullary
    - a. The sp
    - b. Uremic
  - D. Simple ren
  - E. Multilocul
  - F. Miscellane
    - a. Retrope
    - b. Dysont
      - i. Ren
      - ii. End
  - G. Cysts in re
    - a. Pyeloc
    - b. Perical
    - c. Perinep

Table 9. Classifi…

- I. Renal dyspl
  - A. Multicys
  - B. Focal ar
  - C. Multiple struction
- II. Polycystic k
  - A. Infantile
    - 1. Poly
    - 2. Poly
      - a. C
      - b. M
  - B. Adult p
- III. Cortical cys
  - A. Trisomy
  - B. Tuberou
  - C. Simple
    - a. Solit
    - b. Mult
- IV. Medullary
  - A. Medulla
  - B. Medullary cystic diseases
  - C. Medullary necrosis
  - D. Pyelogenic cyst
- V. Miscellaneous intrarenal cysts
  - A. Inflammatory
    - a. Tuberculosis
    - b. Calculus disease
    - c. Echinococcus disease
  - B. Neoplastic—cystic degene
  - C. Traumatic—intrarenal hem
- VI. Extraparenchymal cysts
  - A. Parapelvic
  - B. Perinephric

Table 10. and Ses

1. Autosom
2. Autosom
3. Unilater
4. Solitary
5. Dysplast
6. Pluricyst
7. Juvenile
8. Medulla
9. Glomeru
10. Multiloc
11. Renal cy
12. Renal ly tasia
13. Pyelocal lymphoc
14. Acquire
15. Renal ce

Table 11. The Liapis and W… Classification of Renal C…

- A. Polycystic kidney disease
  - 1. Autosomal-dominant polycystic
    - Classic ADPKD
    - Early onset ADPKD in children
  - 2. Autosomal-recessive polycystic
    - Classic ARPKD in neonates and
    - Medullary duct ectasia in older fibrosis
  - 3. Glomerulocystic kidney disease
    - Familial GCKD
    - Renal hypoplasia and *UROM*
    - Associated with *HNFB1* mutati
    - Hereditary GCKD
    - Associated with ADPKD/ARPKD
    - Syndromic nonhereditary GCKD
    - Sporadic GCKD
    - Acquired GCKD
- B. Renal medullary cysts
  - 1. Nephronophthisis
    - Nephronophthisis, autosomal re
    - Juvenile nephronophthisis
    - NPH1, NPH4
    - NPH1, NPH5 associated with S
    - Infantile NPH2
  - 2. Medullary cystic diseases
    - Autosomal dominant MCKD
    - MCKD associated with hyperur
  - 3. Medullary sponge kidney
- C. Cysts in hereditary cancer syndron
  - 1. von Hippel-Lindau disease
  - 2. Tuberous sclerosis
- D. Multilocular renal cyst
- E. Localized cystic disease
- F. Simple cortical cysts
- G. Acquired (dialysis-induced) cysts
- H. Miscellaneous
  - 1. Pyelocaliceal diverticula
  - 2. Perinephric pseudocysts
  - 3. Hygroma renalis

Table 12. Bonsib (2009) Classification of Renal Cystic Diseases and Congenital Anomalies of the Kidney and Urinary Tract

- I. Polycystic renal diseases
  - A. Autosomal-recessive polycystic kidney disease
    - Classic in neonates and infants
    - Childhood with hepatic fibrosis
  - B. Autosomal-dominant polycystic kidney disease
    - Classic adult form
    - Early onset childhood form
  - C. Acquired renal cystic disease
  - D. Glomerulocystic kidney diseases
  - A. Familial GCKD
    - Renal hypoplasia and *UROM* mutation
    - Associated with *HNFB1* mutations
  - B. Hereditary GCKD
    - Associated with ADPKD/ARPKD/TSC
  - C. Syndromic nonhereditary GCKD
  - D. Sporadic GCKD
  - E. Acquired GCKD
- II. Congenital anomalies of the kidney and urinary tract
  - A. Renal agenesis and dysplasia
    - Agenesis
    - Sporadic: unilateral or bilateral
    - Syndromic
    - Nonsyndromic, multiple malformation syndromes
    - Renal dysplasias
    - Sporadic: unilateral or bilateral
    - Syndromic
    - Nonsyndromic, multiple malformation syndromes
    - Hereditary adysplasia
  - B. Renal hypoplasias
    - Simple hypoplasia: unilateral or bilateral
    - Oligomeganephronic hypoplasia
    - Reduced nephron generations ("cortical hypoplasia")
    - Reduced nephron numbers (premature and low birth weight risk of hypertension)
  - C. Abnormalities in form, position, and number
    - Rotation anomaly
    - Renal ectopias
    - Renal fusions
    - Supernumerary kidney
    - In combination with A, B, or D
  - D. Ureteral and urethral abnormalities
    - Ureteropelvic junction obstruction
    - Ureteral duplication/bifid ureter
    - Vesicoureteral reflux
    - Primary megaureter
    - Ureteral ectopia
    - Posterior urethral valves
    - In combination with A, B, or C
- III. Tubulointerstitial syndromes ±cysts
  - A. Renal tubular dysgenesis
    - Autosomal recessive
    - Secondary twin-twin transfusion
    - ACE inhibitor
  - B. Nephronophthisis: types 1–6
  - C. Medullary cystic diseases:
    - Type 1
    - Type 2/familial juvenile hyperuricemic nephropathy
  - D. Bardet-Biedel syndromes, types 1–12
- IV. Cystic neoplasms and neoplastic cysts
  - A. Cystic nephroma
  - B. Cystic partially differentiated nephroblastoma
  - C. Mixed epithelial and stromal tumor
  - D. Multilocular cystic renal cell carcinoma
  - E. Tubulocystic renal cell carcinoma
  - F. Von Hippel–Lindau disease
  - G. Lymphangioma/hygroma renalis
- V. Miscellaneous cysts
  - A. Simple cortical cysts

Bonsib S. Arch Pathol Lab Med – Vol 134, april 2010

Bosniak Classification:

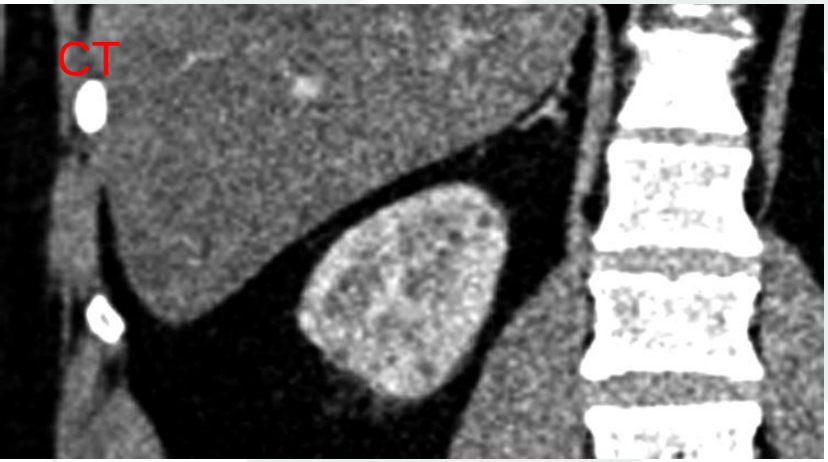
Category (Bosniak)	US Features	Workup
Type 1: Simple cyst	Round, anechoic, thin wall enhanced through transmission	**None**
Type 2: Mildly complicated cyst	Thin septation, calcium in wall	**CT or US follow-up**
Type 3: Indeterminate lesion	Multiple septae, internal echos mural nodules Thick septae	**Partial nephrectomy, biopsy** **CT follow-up if surgery is high risk**
Type 4: Clearly malignant	Solid mass component	**Nephrectomy**

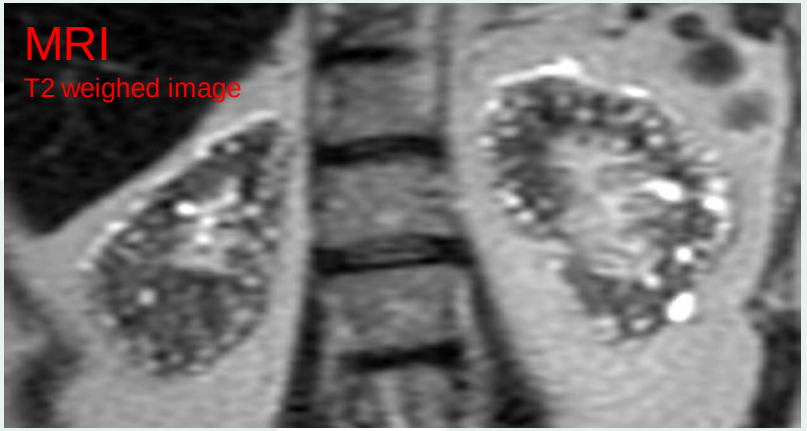
Based on the CT graphic appearance of renal cysts

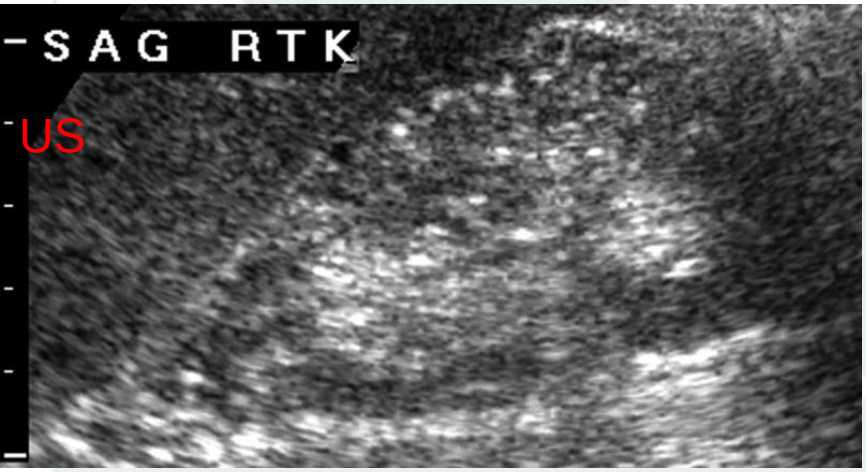
Acquired cystic kidney disease

- Dialysis patients
- Non enlarged kidneys
- Despite multipe cysts
- Cysts of different size
- Hyperechogenic parenchyma irt liver/spleen
- Distal wall enhancement

Lithium nephropathy: sonographic findings



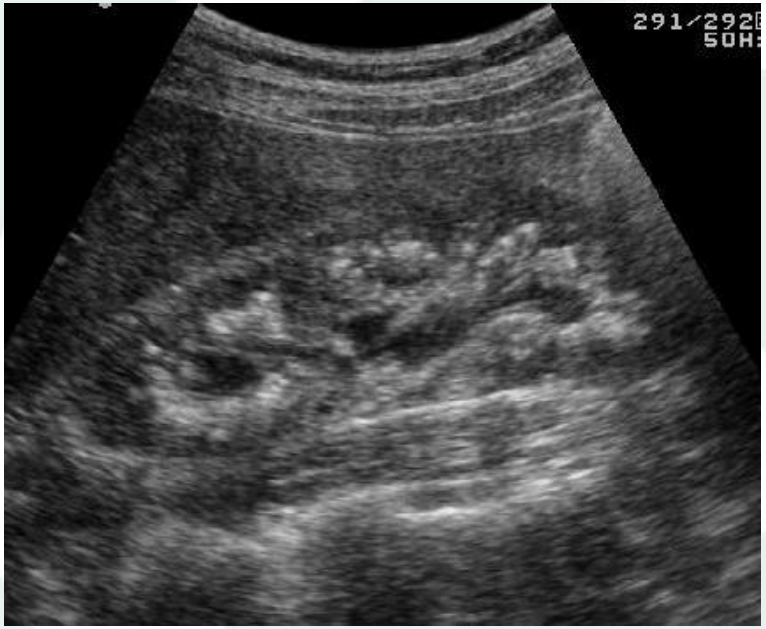


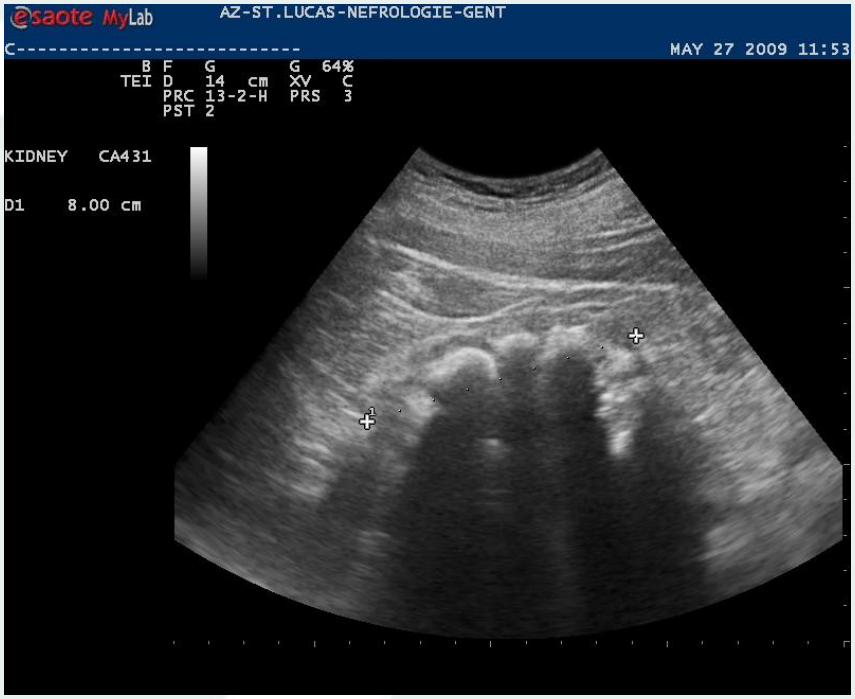


- Lithium-> nephrogenic DI, RTA, TI nephritis, nephrotic S
- Observational series of 10 ptn/36.000 US exams/4 years)
- **Numerous microcysts & punctate echogenic foci (microcalcifications, predominantly in the cortex)**
- Punctate echogenic foci better seen on US than on CT and MRI
- Kidney size : 7.5-13.9 cm (mean 11.2 cm)
- Probably rather rare finding
  Unknown how many lithium ptns who don't show these findings

DM di Salvo et al. J Ultrasound Med 2012; 31/ 637-644

Medullary sponge kidney (Cacci Ricci)





Medullary calcifications

Reversed CM differentiation

Medullary sponge kidney (MSK) is a developmental abnormality occurring in the medullary pyramids of the kidney. MSK is characterized by cystic dilatation of the collecting tubules in 1 or more renal pyramids in 1 or both kidneys **(**asymptomatic/kidney stones/hematuria/UTI)

Nephrocalcinosis

- Deposits of CaP (& Caox) in renal parenchyma and tubuli
- Nephrocalcinosis involves the renal medulla or, much less often, the cortex
- « Reversed corticomedullary differentiation »
- Sometimes with retro-acoustic shadow
- Not specific for 1 condition

Diseases associated with nephrocalcinosis

- 30-40% Hyperparathyreoidism
- 20% Renal tubular acidosis
- 10-15% Medullary sponge kidney
- 6% Hypercalciuria, idiopathic
- 5% End stage renal disease
- rest Sarcoidosis, malign tumors, Vitamin D intox, Papillary necrosis…

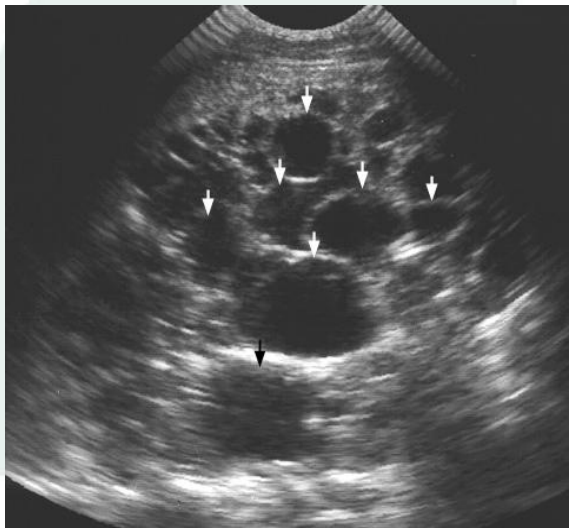
Autosomal dominant polycystic kidney disease

- Diagnosis can be made by US alone
- Positive family history and:

15-30 years: 2 cysts in 1 kidney or 1 cyst in each kidney

30-60 years: 2 cysts in each kidney

> 60 years: 4 cysts in each kidney



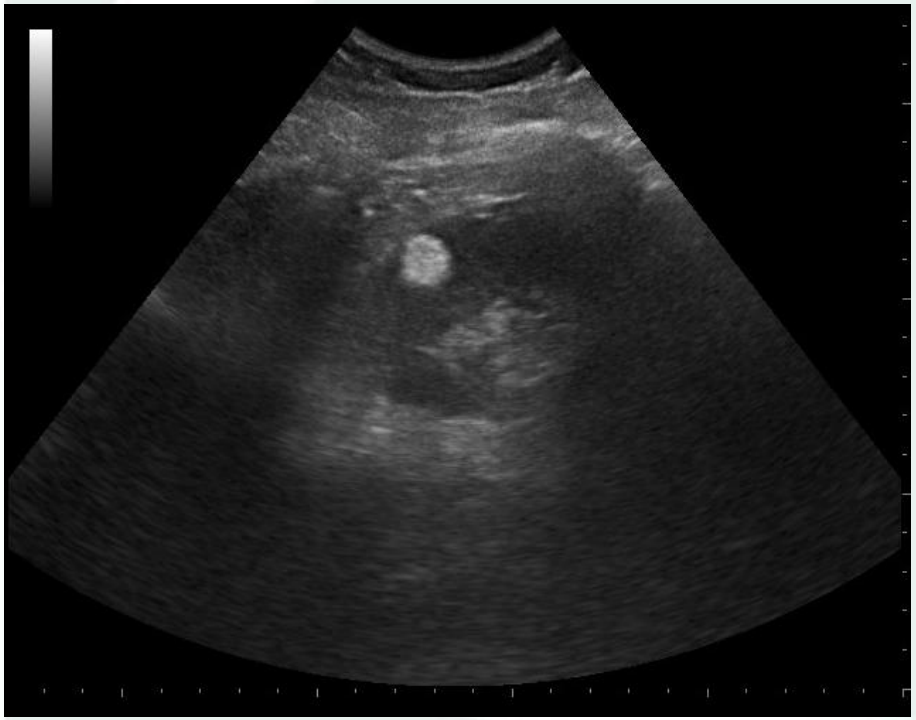
Renal tumours

- Benign vs malignant
- Sensitivity of US to detect tumour depends on its size
- Tumours > 3 cm : in 100% detected
- Tumours < 2 cm: in 50% detected

Benign renal tumours

- Angiomyolipoma
- Oncocytoma
- Multilocular cystic nephroma

Angiomyolipoma



- Most frequent benign kidney tumour
- Autopsy studies : 0.3 %
- Female/male: 3/1
- 95% fat
- Round hyperechogenic, « white » tumor
- Usually rather small (1 cm)
- No blood flow detectable with CF mode
- 1/3 : retro-acoustic shadow
- Check after 3 months -> idem aspect: diagnosis is confirmed

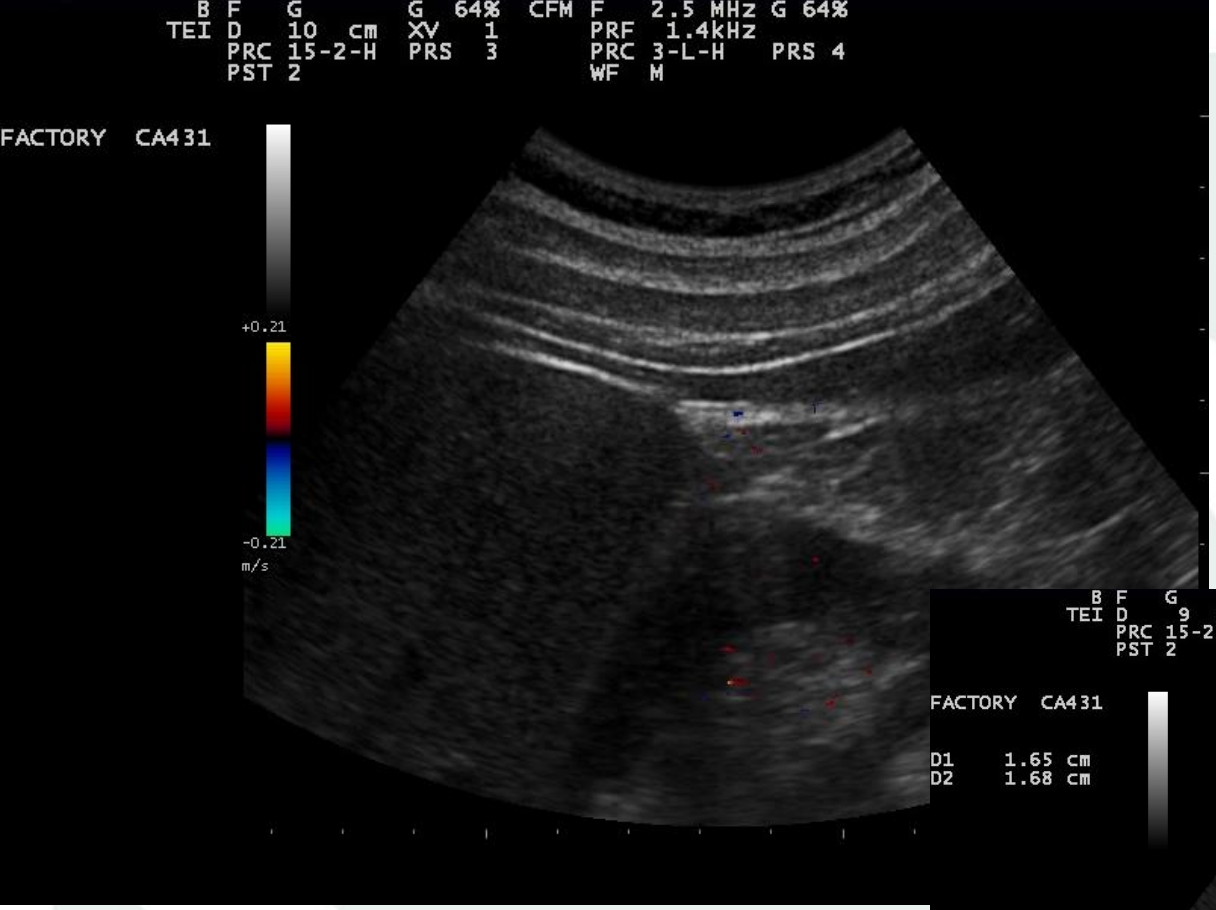
If bilateral :
Think of tuberous sclerosis (Bourneville)

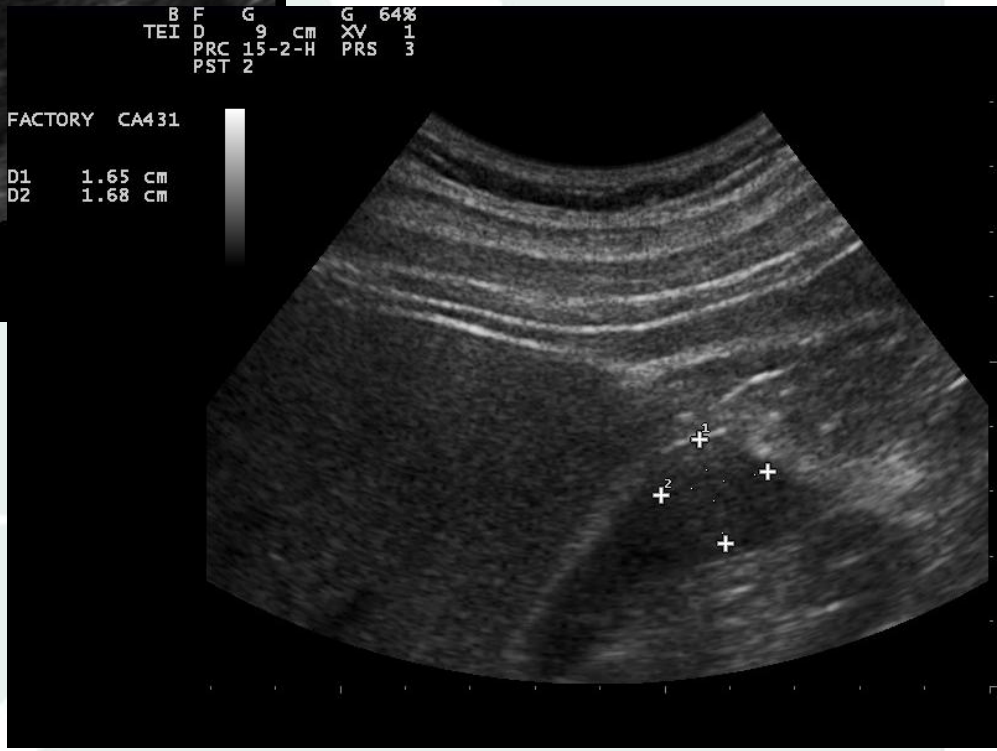
Malignant renal tumours

- RCC (80-85%)
- TCC (8%)
- Lymphoma
- Metastases
- Other: f ex sarcoma, …

Renal Cell Carcinoma

- 80 % of the malignant renal tumors
- APO : clear cell – papillary - chromophobe
- Frequently hyperechogenic
- But can also be hypo or iso echogenic
- Color doppler: hypervascularisation with low RI
- Central necrosis
- 5% Multilocular cystic aspect





Papillary renal cell carcinoma
R lower pole

No vascularisation shown with colordoppler, doens't exclude malignancy !

Thank you for your attention !