***Renal parenchymal neoplasm***

***Benign tumor***

1-**Renal adenoma**

-most common benign renal parenchymal lesion.

-These are small well differentiated glandular tumor of renal cortex.

-they are usually asymptomatic & discovered incidentally

-No clinical, histological, or immune histochemical criteria differentiate adenoma from carcinoma.

-renal tumors less than 3cm usually were considered adenoma and had little propensity for metastasis

2-**Renal oncocytoma**

-Has spectrum of behavior ranging from benign to malignant.

-Composed of large epithelial cell with eosinophilic cytoplasm (oncocyte cell).

-Gross hematuria or flank pain occur in less than 20% of patients.

-No characteristic features of the tumor appear on CT, U/S, IVU, or MRI.

-Angiographic features including (spoke wheel) appearance of the arterioles.

3-**Angeomyolipoma(AML) or renal hamertoma**

-They are characterized by 3 histologic component fat cell, smooth muscle & blood vessels.

-Usually (about 45-80% of AML) associated with tuberous sclerosis.

-Negative density -20 to -80 hounsfield units in CT pathognomonic for AML.

-Treatment depend on

1-size

2-tuberous sclerosis

3-size of lesion by CT (4cm more or less)

4-**Other rare tumor like**

-leiomyoma,

-hemangioma,

-renal lipoma &

-juxtaglomerular cell tumor (renin secreting tumor) which is always benign

***Adenocarcinoma of the kidney***

***Renal cell ca***

-most commonly in the 5th-6th decade (m:f ratio 2:1)

-The cause of RCC remain unknown.

-It originate from the proximal convoluted tubule of the cortex & tend to grow out into the perinephric tissue.

Histologically

-most often mixed adenocarcinoma containing clear cells, granular cells, and occasionally, sarcomatoid appearing cells

RCCs are vascular tumors

**Spread**

* direct invasion through renal capsule
* direct extension into the renal vein.

-25-30% of patient have evidence metastatic disease

at presentation.

**Risk Factores**

1-smoking is only definitive risk factor

2-occupational

3- genetic

4-Acquired renal cystic disease

***Tumor grading & staging***

The ultimate goal of staging is to select appropriate therapy & obtain prognostic information.

Stage 1—tumor is confined within the renal parenchyma.

Stage 2—tumor is confined within the gerota fascia

 (including perinephric fat & adrenals).

Stage 3a—tumor involve main renal vein or IVC.

 3b—tumor involve regional LN.

 3c—tumor involve both local vessel & regional LN.

Stage 4a—tumor involve adjacent extragerotal organs

 (colon, pancreas, etc).

Stage 4b—distant metastases.

Grading are 4 grades from well differentiated to undifferentiated

***Symptoms & signs***

1-The classical triad of gross hematuria, flank pain, &palpable mass occur in 7-10 % of patients & frequently manifestation of advance disease.

2-60% of pt present with gross or microscopic hematuria.

3-Pain abdominal mass or both occur in 40% of pt.

4-Symptoms secondary to metastases dyspnea, cough, seizure, headache, or bone pain.

-Renal tumor increasingly discovered incidentally due to the use of CT more than 50%

**Paraneoplastic syndrome**

* occur in 10-40% of RCC patient.
* It include erthrocytosis, hypercalcaemia, hypertension and non metastatic hepatic dysfunction
* It does not indicate apoor prognosis
* Usually relieve after nephractomy

Laboratory finding

1-Anemia 30%

2- increse ESR.

Imaging

U/S : it highly accurate in distinguishing simple cyst from solid lesion

CT :

* It more sensitive than U/S and IVP
* show renal mass that ***enhanced*** with contrast
* CT is method of staging

MRI :to evaluate vascular invasion

Angiography

Radionuclide imaging

***Management***

Mainly depend on the stage of the tumor

***Localized***—**Radical nephrectomy** when kidney, perirenal fat & adrenal gland removed .

***Disseminated***—30% of pt present with metastases usually aggressive & rapidly progressive.

* Palliative surgery
* Radiotherapy,
* Hormonal therapy,
* Chemotherapy & biologic response modifier like interferon & interleukin
* Observation.

**Notce** prognosis is mainly depend on performance state

***Nephroblastoma***

***(Wilms tumor)***

* Most common solid renal tumor of childhood
* peak age for presentation is the 3rd year of life
* there is no sex predilection
* 10% have congenital malformation like aniridia and genitourinary abnormalities.

***Pathology***

* Tumour precursor lesion is **nephrogenic rests**
* **T**ypically consist of ***blastemal,epithelial,***and ***stromal***
* **It of 2 types :*Favorable*** contain no **anaplasia** and

***Un favorable*** containing **anaplasia**

**Metastases**

 **D**iract **H**ematogenouse **L**ymphatic

***Clinical finding***

* Asymptomatic mass is the most common presentation discovered by the family member or physician.
* abdominal pain, distension, nausea, vomiting, anorexia, fever.
* The most common sign is abdominal mass
* Hypertension
* Hematuria

laboratory—hematuria & anemia

***Imaging***

**U/S**—is the current initial study of choice to evaluate palpable abdominal masses.

**CT**—useful in providing tumor extension, state of contralateral kidney &LN involvement.

**IVU**—to evaluate renal masses, but had been replaced by newer modality.

**Chest x-ray**—to evaluate the presence of lung metastases

\*Needle biopsy—indicated if

* Tumor too large for resection
* For which chemotherapy or radiotherapy is planned.

***D.Dx***

Hydronephrosis.

Cystic kidney.

Neuroblastoma.

***Treatment***

1-surgical measure, radical nephrectomy

2-chemptherapy, wilms tumor is chemosesitive.

3-radiotherpy, its also radiosensitive