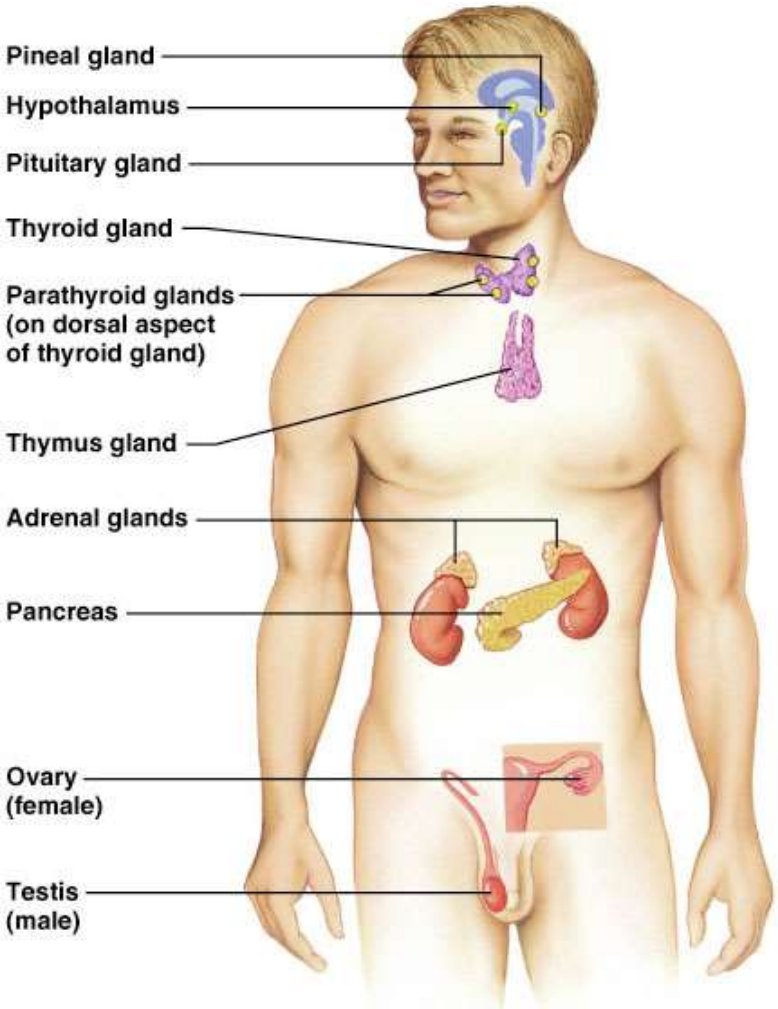
NEUROENDOCRINE SYSTEM

Nuclear Medicine Scans: Analysis of uptake mechanism and imaging protocols

NEUROENDOCRINE SYSTEM



MEN	GENE
MEN 1	MEN1-TSG
MEN 2 (A, B)	RET
FMTC	RET
von Hippel-Lindau Sy.	VHL
von Recklinghausen Sy.	NF-1
Carney Sy.	PRKAR-1A
Cowden Sy.	PTEN
McCune–Albright Sy.	GNAS-1

NEUROENDOCRINE SYSTEM

The Endocrine system is divided into :

- Endocrine organs dedicated to production of hormones e.g. pituitary,thyroid….etc
- Endocrine components in clusters in organs having mixed functions e.g. pancreas, ovary, testes…..
- Diffuse endocrine system comprising scattered cells within organs acting locally on adjacent cells without entry into blood stream

PITUITARY GLAND

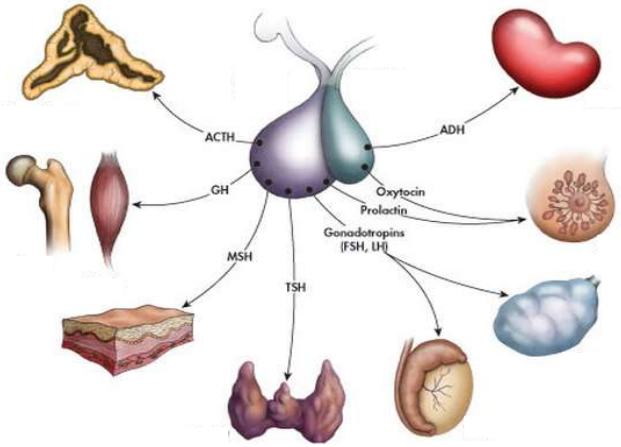
- Pituitary in sella turcica,& weighs about 0.5gm.
- Connected to the HYPOTHALAMUS with stalk.
- Composed of :

A-ADENOHYPOPHYSIS- (80%)

- Blood supply is through portal venous plexus
- Hypothalamic-Hypophyseal feed back control

B- NEUROHYPOPHYSIS

- From floor of third ventricle
- Modified glial cells & axons hypothalamus.
- Has its own blood supply.



PITUITARY GLAND

A - Anterior pituitary (Adenohypophysis)

1-Somatotrophs from acidophilic cells → Growth H.
2- Lactotrophs from chromophobe cells → Prolactin
3- Corticotrophs from basophilic cells → ACTH,MSH .
4- Thyrotrophs from pale basophilic cells → TSH
5- Gonadotrophs from basophilic cells → FSH, LH

B - Posterior pituitary (Neurohypophysis)
1- Oxytocin
2- ADH

Disease divided into :

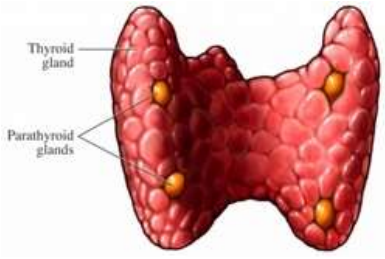
1- Diseases of overproduction of secretion
(Hyperfunction)
2- Diseases of underproduction
(Hypofunction)
3- Mass effects (Tumors)

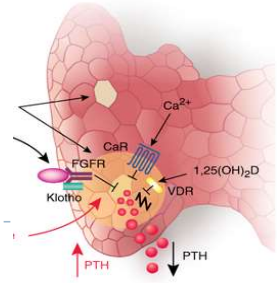
N.B. Correlation of clinical picture , hormonal assays , biochemical findings , together with pathological picture are of extreme importance in most conditions.

PARATHYROID GLAND

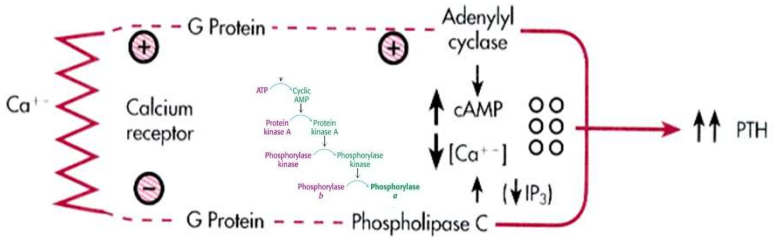
- Derived from the third and fourth pharyngeal pouches.
- 90% of people have four glands.
- Location: mostly close to the upper or lower poles of the thyroid.
- Can be found anywhere along the line of descent of the pharyngeal pouches.
- There are two types of cells with intervening fat :

  - Chief & Oxyphil cells
- Secretion of PTH is controlled by level of free calcium Ca_2^+ = 1,9mmol/l (7.8mg/dl) 5x ↑PTH

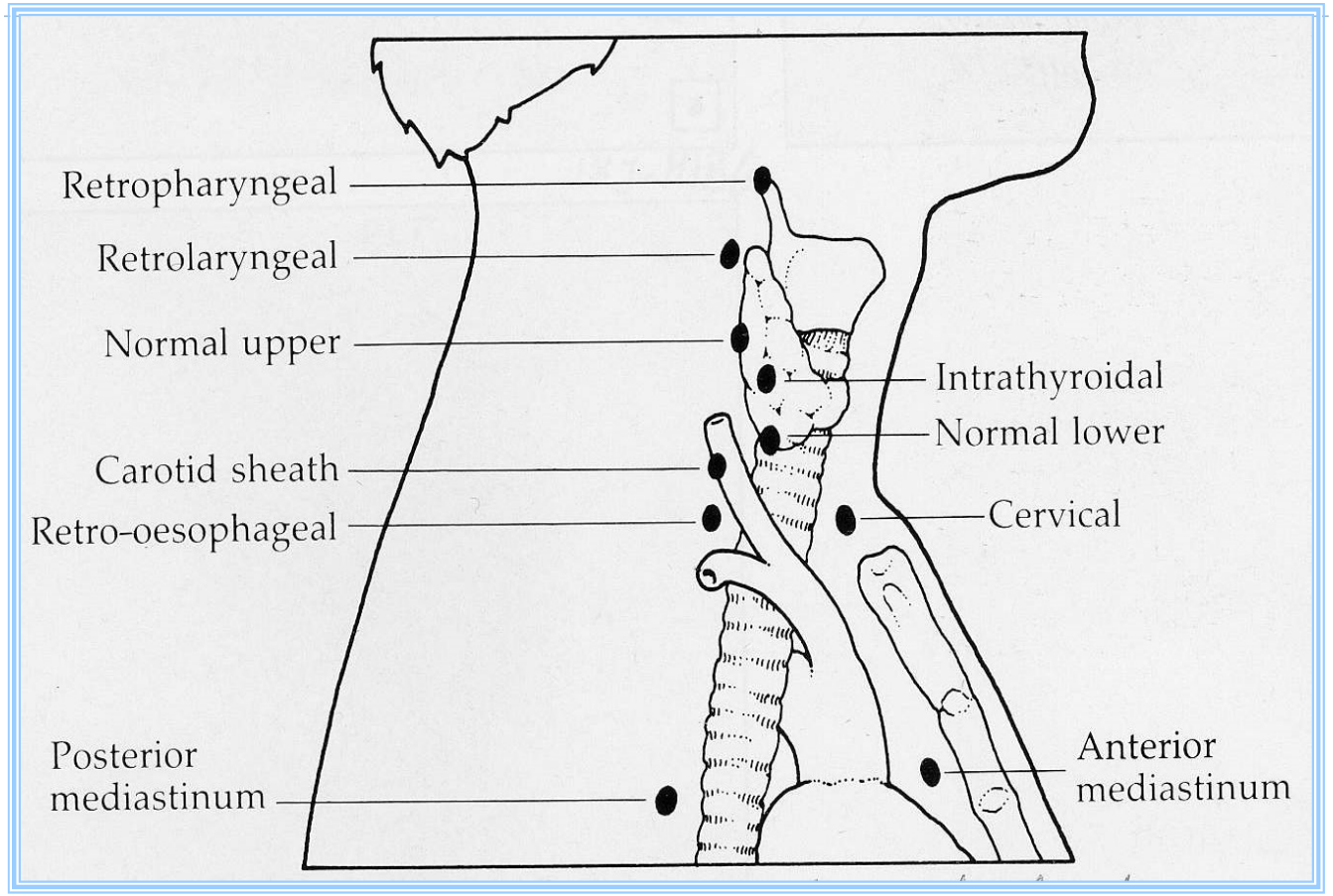




[Ca⁺⁺] Low

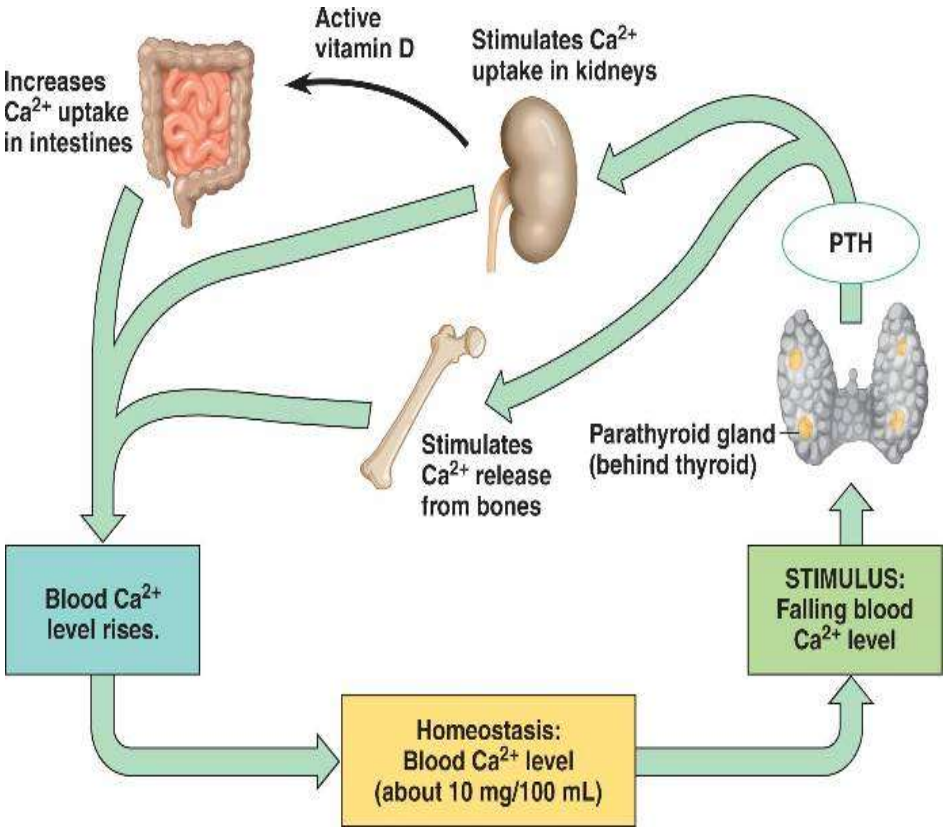


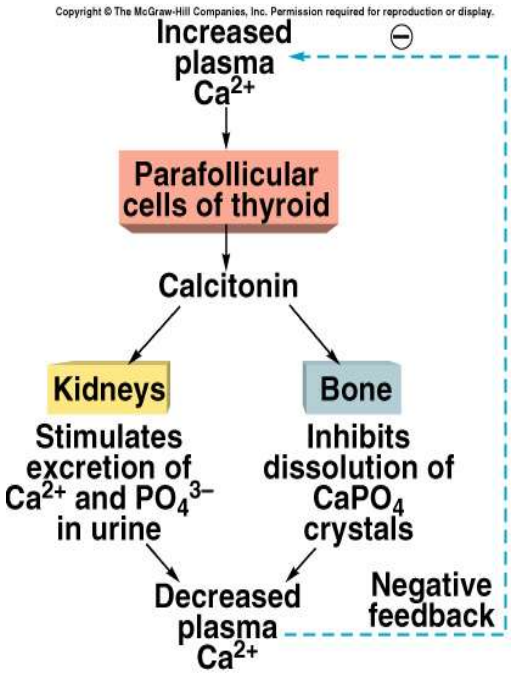
PARATHYROID GLAND

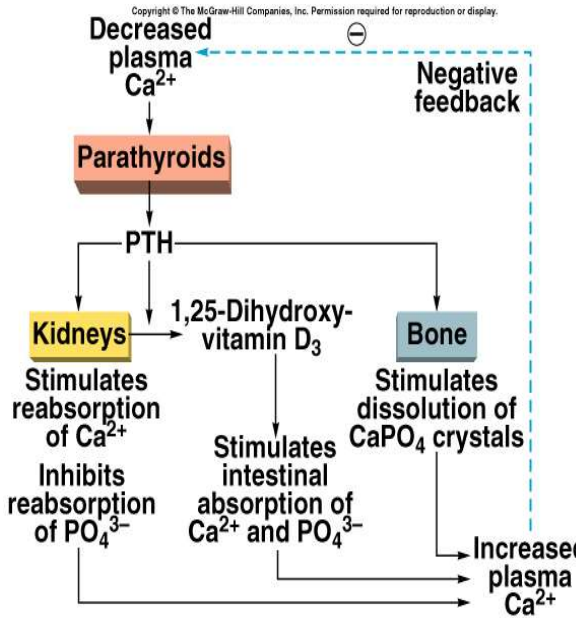


Williams textbook of endocrinology

PTH Ca TURNOVER





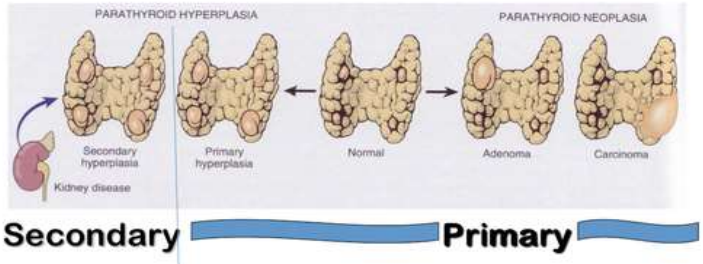


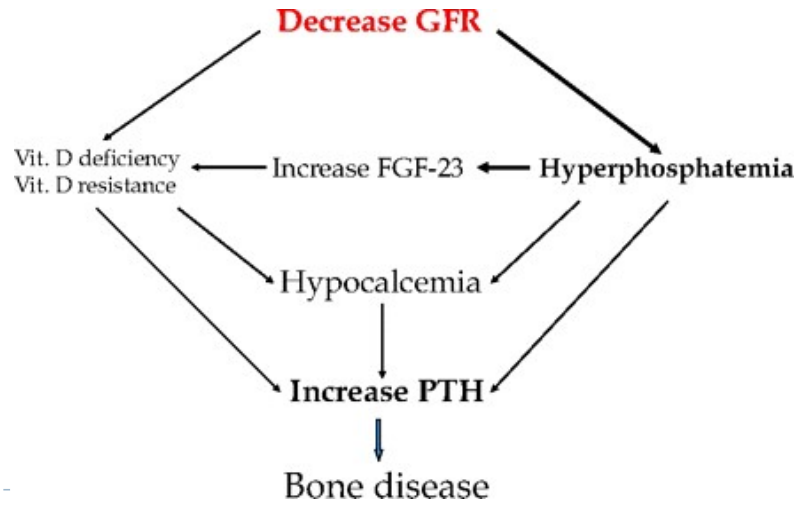
- Williams textbook of endocrinology

Hyperparathyroidism : Primary OR Secondary

Primary Hyperparathyroidism:

- Commonest cause of asymptomatic hypercalcemia
- Female:Male ratio = 2-3 : 1.
- Causes : Adenoma 75%-80%
  Hyperplasia 10-15%
  Carcinoma < 5%
- Majority of adenomas are sporadic
- 5% familial associated with MEN-1 or MEN-2A





Morphology in other organs:

- **Skeletal system:**
  - Bone resorption by osteoclasts, with fibrosis, cysts formation and hemorrhage →Osteitis Fibrosa Cystica
  - Collections of osteoclasts form ' Brown Tumors"
  - Chondrocalcinosis and pseudogout may occur.

- **Renal system**:
  - Ca. Stones. & Nephrocalcinosis.

- **Metastatic calcification in other organs:**

  Blood vessels & myocardium , Stomach, Lung ...etc

Hyperparathyroidism, clinical picture

- 50% of patients are asymptomatic.
- Patients show ↑ Ca & ↑PARATHORMONE levels in serum
- Symptoms and signs of hypercalcemia:

  Musculoskeletal, Gastrointestinal tract, Urinary

  and CNS symptoms
- Commonest cause of silent hypercalcemia .
- In the majority of symptomatic hypercalcemia commonest cause is wide spread metastases to bone

DIAGNOSIS

Ca
PO_4
PTH
$1,25(OH)_2D3$

- Biochemical findings :
  ↑PTH , ↑ Ca , ↓ phosphate ,↑alkaline phosphatase
- In other causes of hypercalcemia, PTH is ↓

Study Type	Sensitivity	Specificity
Ultrasound[7,10]	71-80%	80%
Endoscopic Ultrasound[10]	71%	
CT Scan[6,7,17,19]	46-80%	88-98%
MRI Scan[6,7,24,28]	64-78%	88-95%
Thallium-Technetium Scan[7,8,16,18]	**75%**	**73-82%**
Technetium-Sestamibi Scan[3, 8,16,18]	**90.7%**	**98.8%**
PET Scan[49,51,53,54]	80-94%	
Angiography & Venous Sampling[7]	91-95%	96-98%
Venous SamplingAlone[11,16,55]	70-80%	

Parathyroid Localization and Implications for Clinical Management

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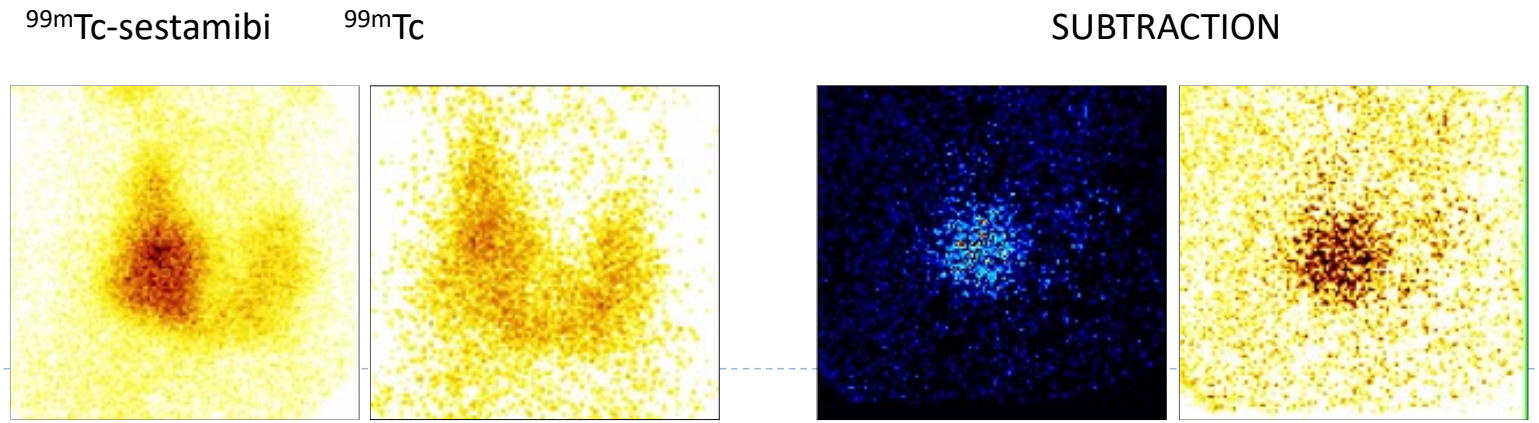
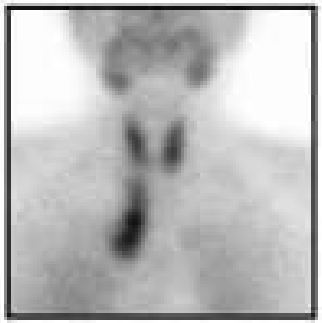
J Clin Endocrinol Metab, March 2013, 98(3):902–912

PARATHYROID SCAN

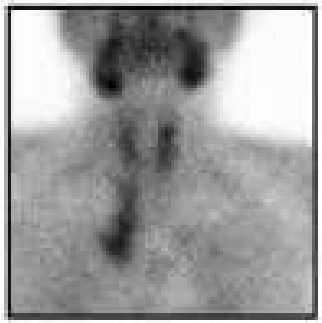
- Consecutive application of two radioactive isotopes 99mTc and 99mTc-MIBI (methoxy isobutyl isonitrile).
- 99mTc binds and shows the thyroid gland, and the excess seen on scintigraphy after subtraction of technetium from 99mTc MIBI, represents only parathyroid tissue.
- Sensitivity: Adenoma 89%; Hyperplasia 66%



PARATHYROID SCAN

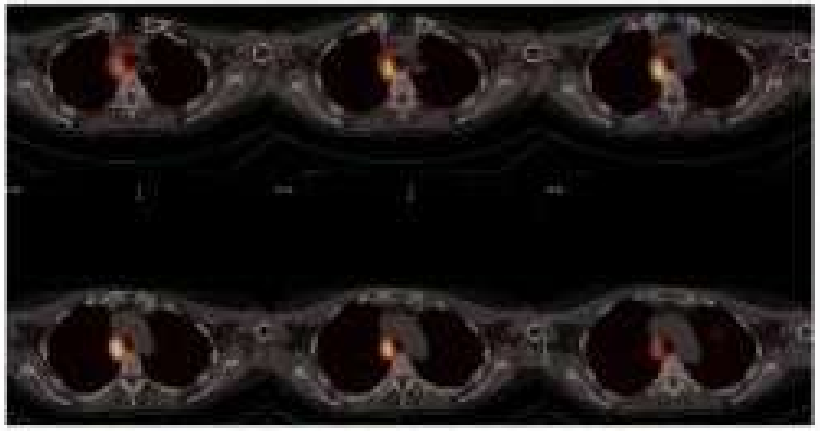


^{99m}Tc-Sestamibi
15 min post-injection

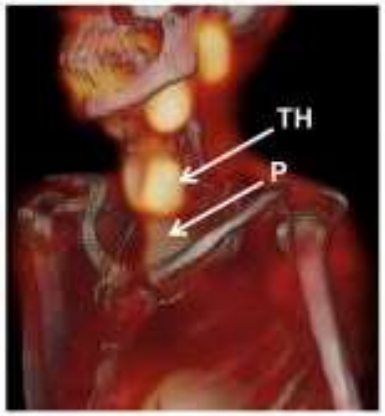


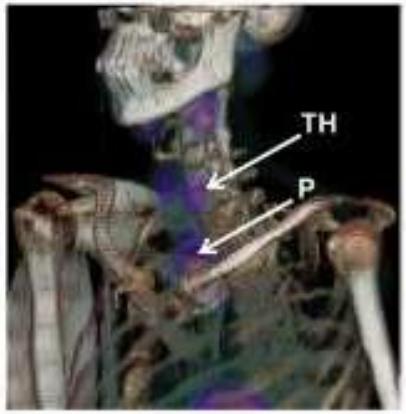
^{99m}Tc-Sestamibi
2.5 h post-injection

$^{99m}TcO_4^-$



Transaxial
SPECT/CT

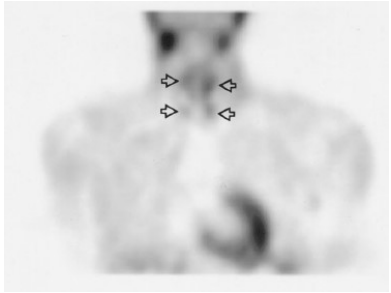


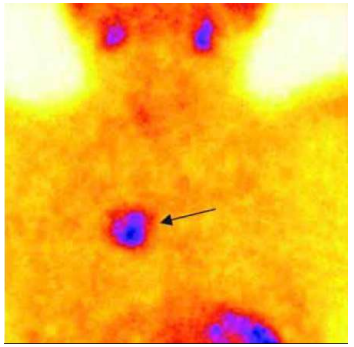


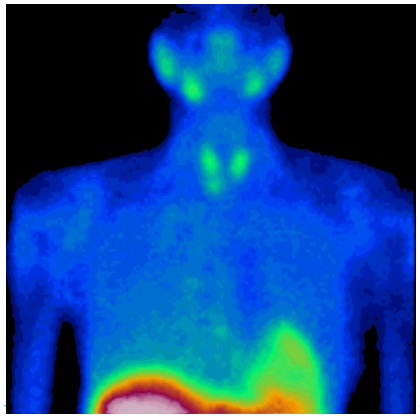
3D volume rendering of SPECT/CT

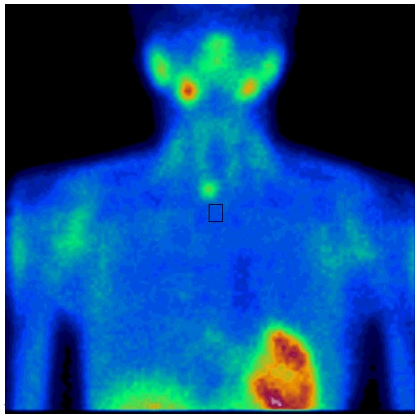
PARATHYROID SCAN

- Two-phase method: early and delayed
- PTG has a 60% slower "washout"
- Extrathyroid localization
- Adenoma sensitivity: Early phase 10 min. – 79% Late phase 2-3h. - 90%





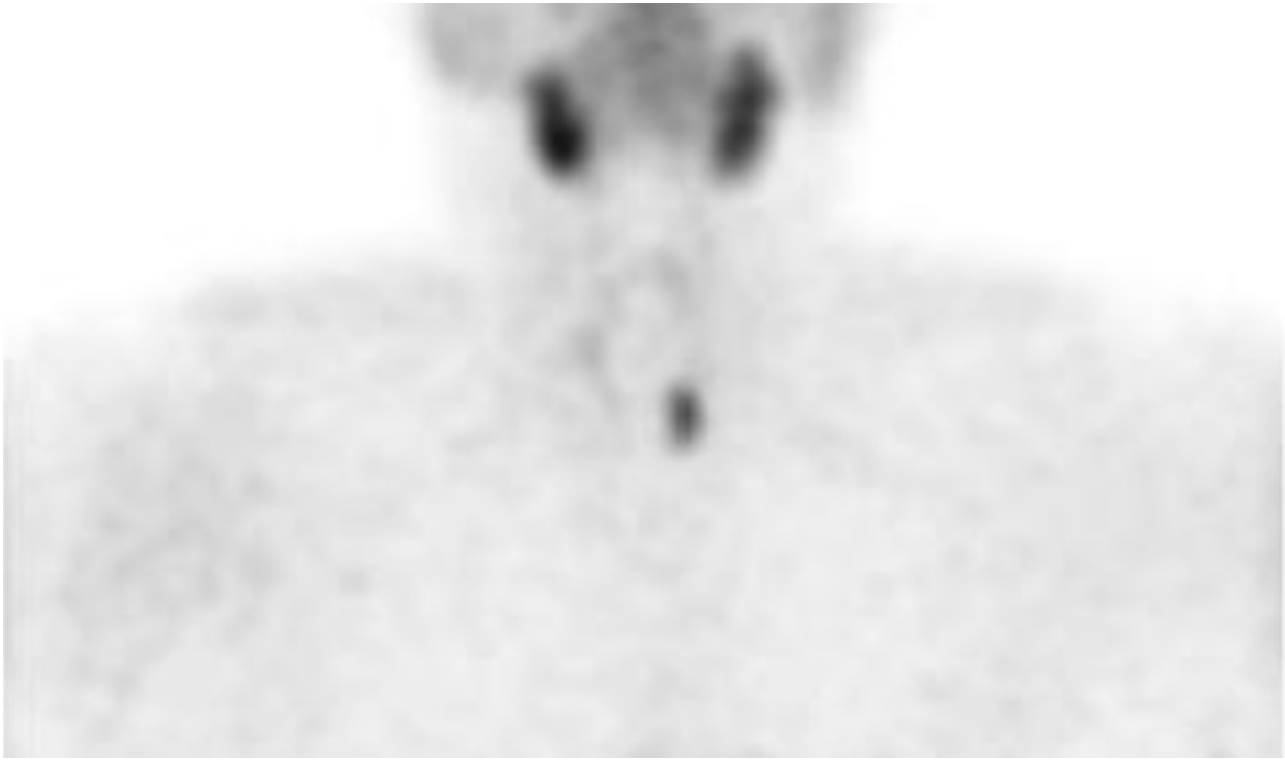




PARATHYROID SCAN

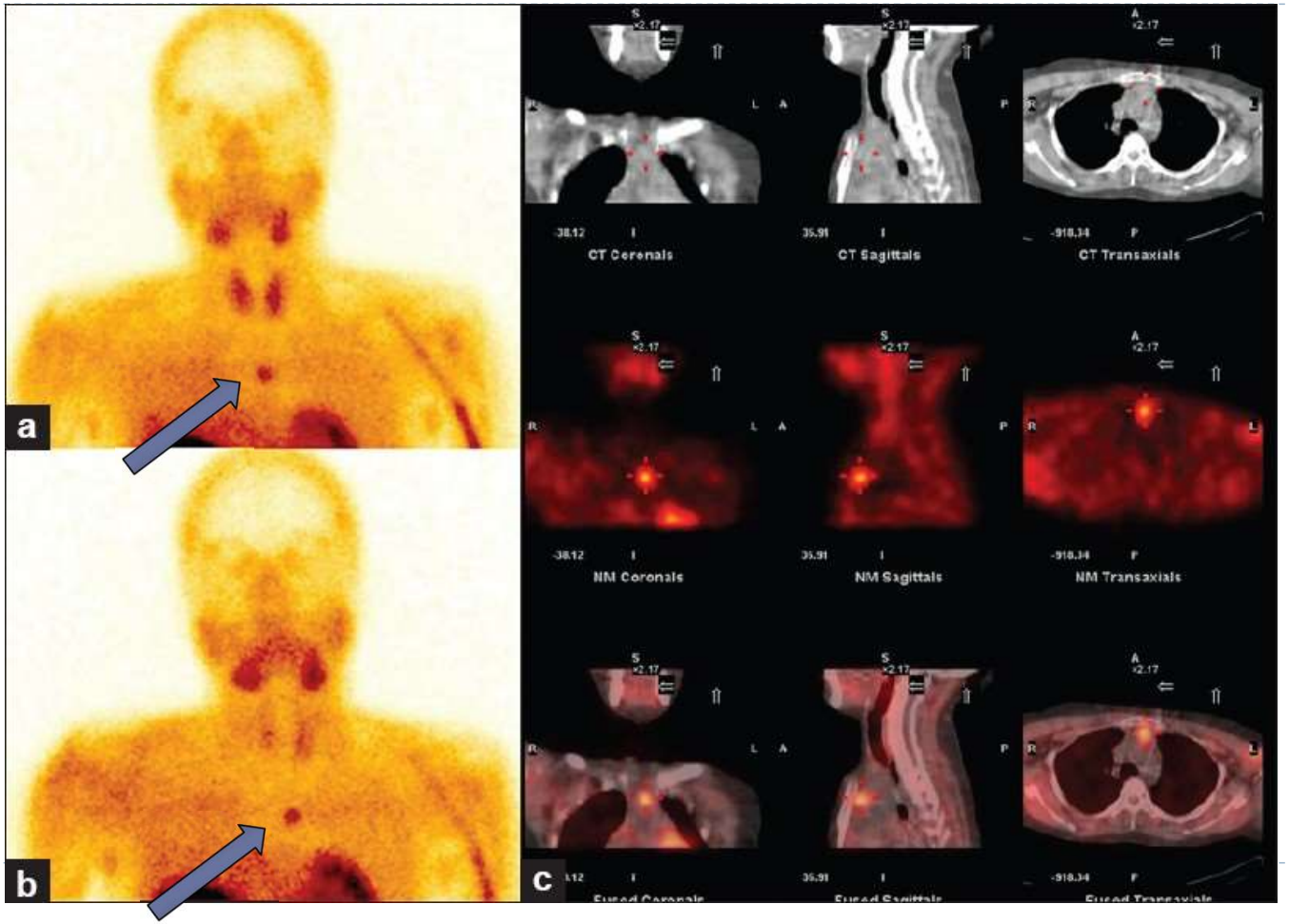
^{99m}Tc-MIBI 10min

PARATHYROID SCAN



^{99m}Tc-MIBI 2h

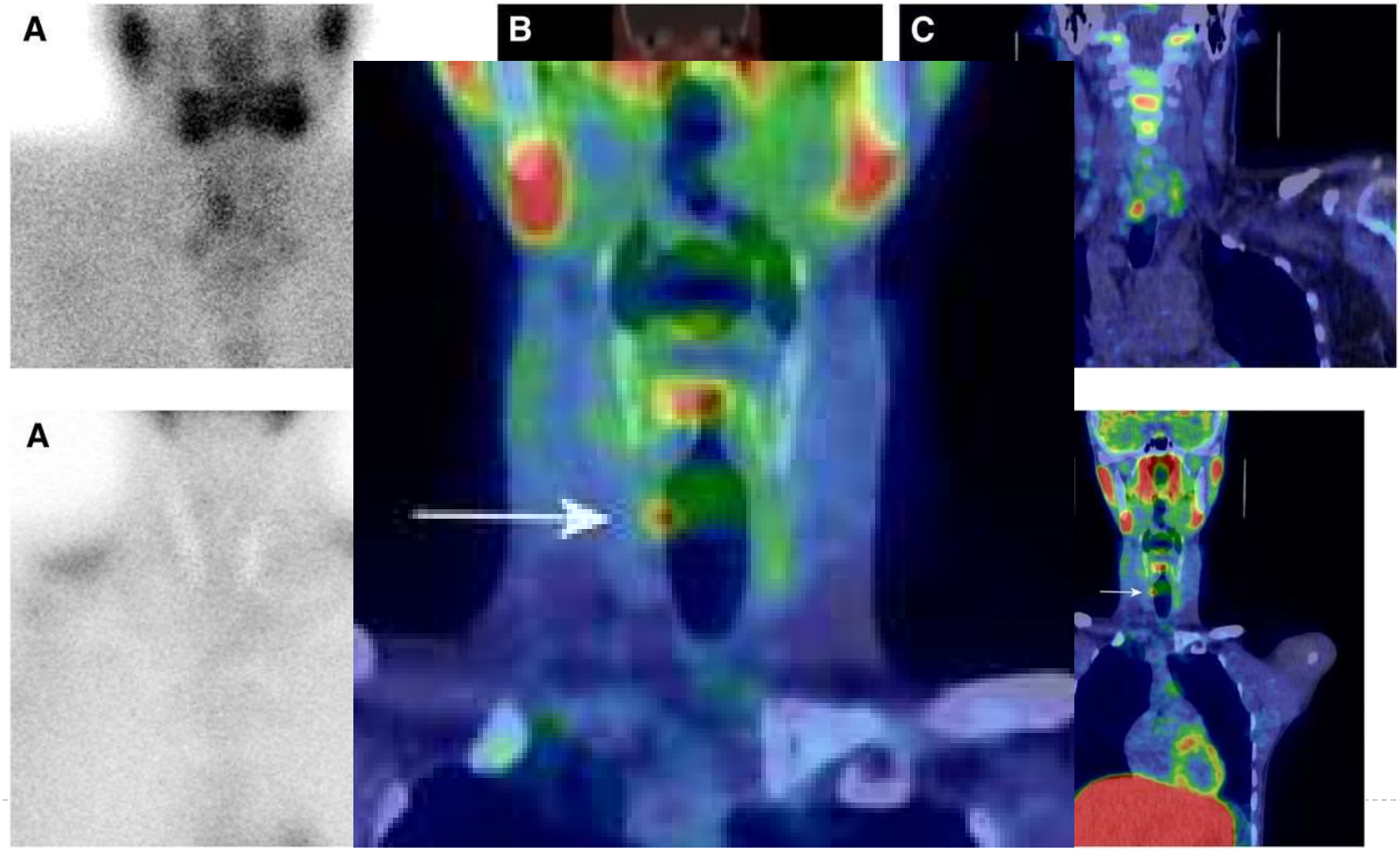
SPECT/CT ^{99m}Tc-MIBI



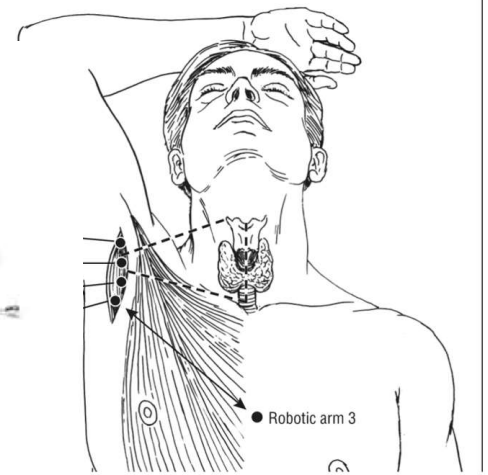
^{99m}Tc/^{99m}Tc-sestamibi

^{99m}Tc-sestamibi SPECT/CT

^{11}C-methionine PET/CT



MINIMALLY INVASIVE PARATHYROIDECTOMY



ADRENAL GLAND

- Weight of normal gland is 4 g.

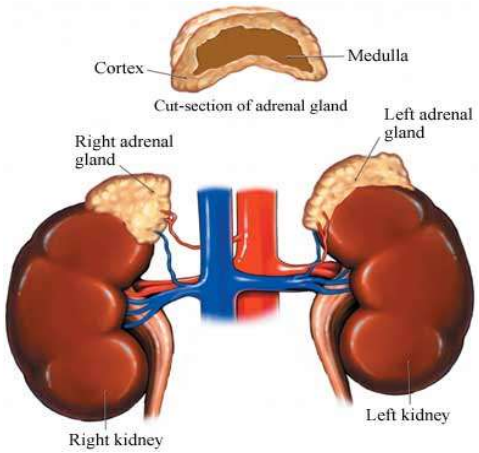
  Adrenal Cortex - Derived from mesoderm & composed of

  1- Zona glomerulosa→mineralocorticoids (aldosteron)

  2- Zona fasciculata → glucocorticoids (cortisol)

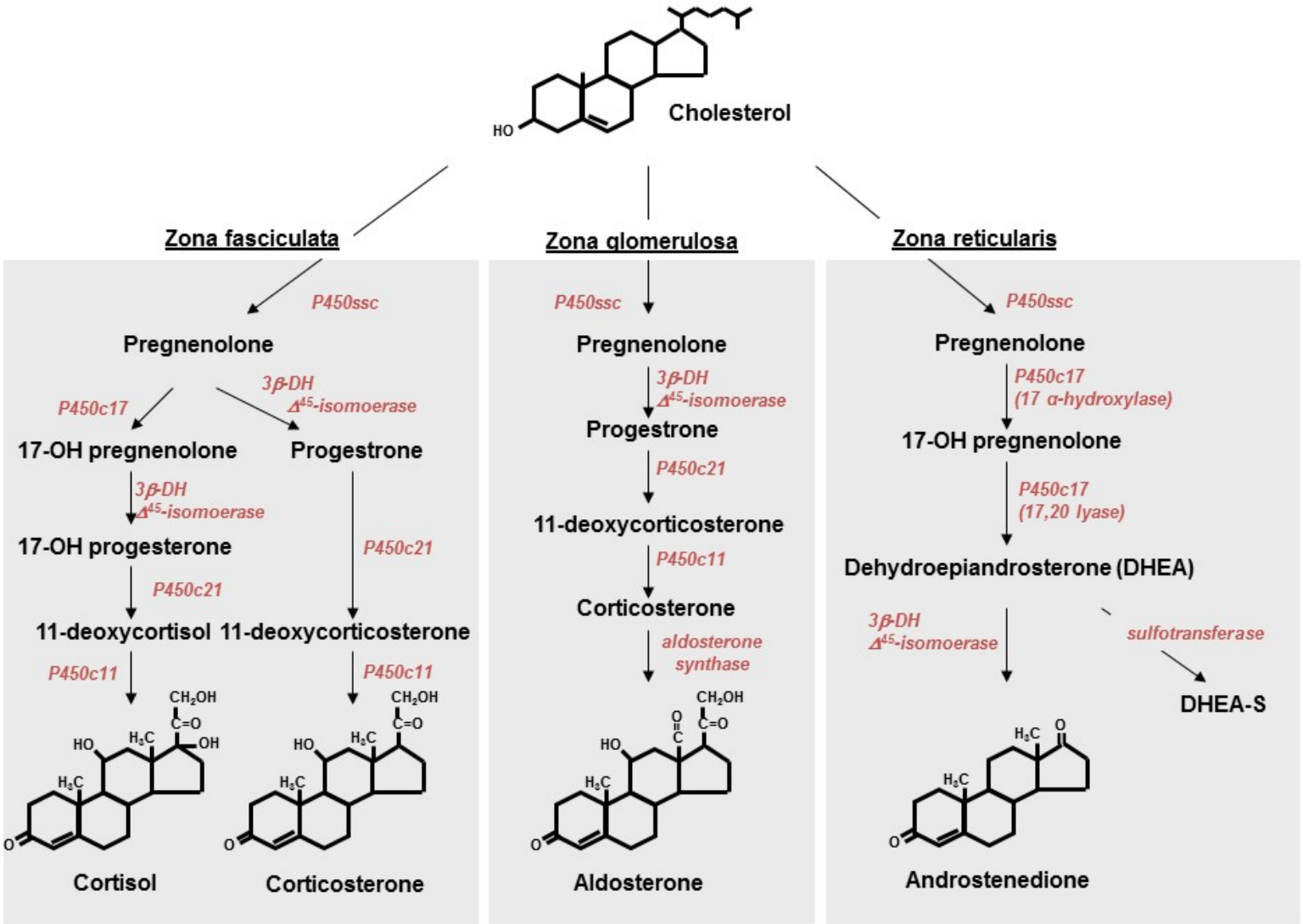
  3- Zona reticularis → estrogens & androgens

- Diseases are those of hyperfunction & hypofunction & tumors



Adrenal Medulla –

- ❖ Derived from neural crest & is part of sympathetic system.
- ❖ Composed of Chromaffin cells secreting catecholamines
- ❖ Diseases are mainly tumors


Cholesterol
Zona fasciculata
Zona glomerulosa
Zona reticularis
P450ssc
Pregnenolone
3β-DH
Δ45-isomoerase
P450c17
17-OH pregnenolone
Progestrone
3β-DH
Δ45-isomoerase
17-OH progesterone
P450c21
P450c21
11-deoxycortisol
11-deoxycorticosterone
P450c11
P450c11
Cortisol
Corticosterone
P450ssc
Pregnenolone
3β-DH
Δ45-isomoerase
Progestrone
P450c21
11-deoxycorticosterone
P450c11
Corticosterone
aldosterone synthase
Aldosterone
P450ssc
Pregnenolone
P450c17
(17 α-hydroxylase)
17-OH pregnenolone
P450c17
(17,20 lyase)
Dehydroepiandrosterone (DHEA)
3β-DH
Δ45-isomoerase
sulfotransferase
DHEA-S
Androstenedione

ADRENOCORTICAL ABNORMALITIES:

- There are 3 syndromes associated with **hyperfunction**:
  - 1- Cushing's Syndrome & Cushing's Diseasr
  - 2- Conn's Syndrome & Hyperaldosteronism
  - 3- Adrenogenital Syndrome
- **Adrenal tumors**
- Encapsulated , usually yellow
- Size variable 1-2 cm. (30gms.)Up to large tumors
- Most incidental nonfunctioning tumors, may be functioning
- Malignant tumors with necrosis, hemorrhage (≥ 300gms)
- Usually larger, more aggressive in adults
- Both may show same appearance of uniform or slightly pleomorphic cells ,may be eosinophilic or clear

ADRENAL SCAN

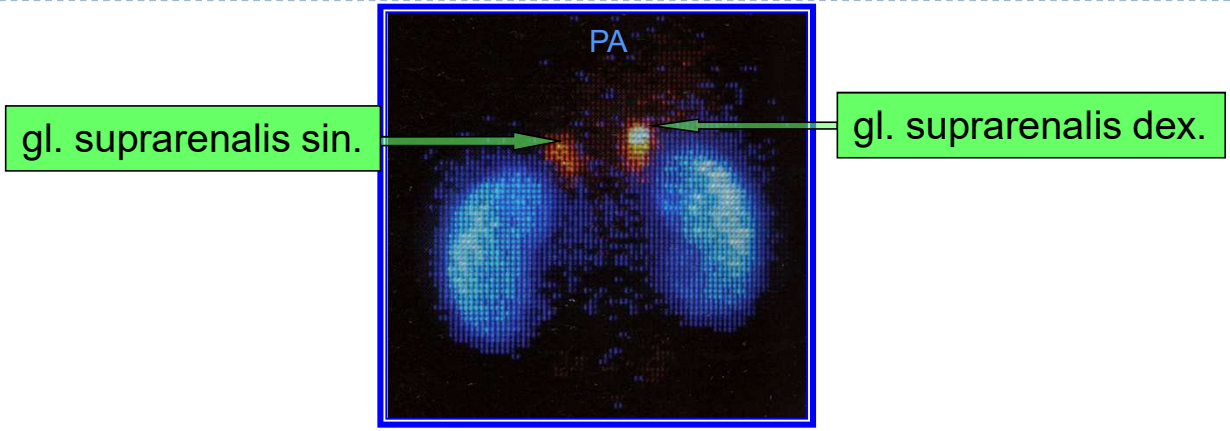
$^{131/123}$I-NP59 (6-beta jodometil 19 norholesterol)

Cholesterol is a substrate for the synthesis of steroid hormones.

The radionuclide-labeled cholesterol analog, 123I-NP59, is incorporated into LDL and thus transported through the circulation. It enters the adrenal cortex using LDL receptors.

It is esterified and stored in intracellular fat droplets, but is not further metabolized. NP59 transported via hepatic LDL receptors is metabolized to fatty acid analogs, and excreted via bile (high RA in the gut on scintigrams).

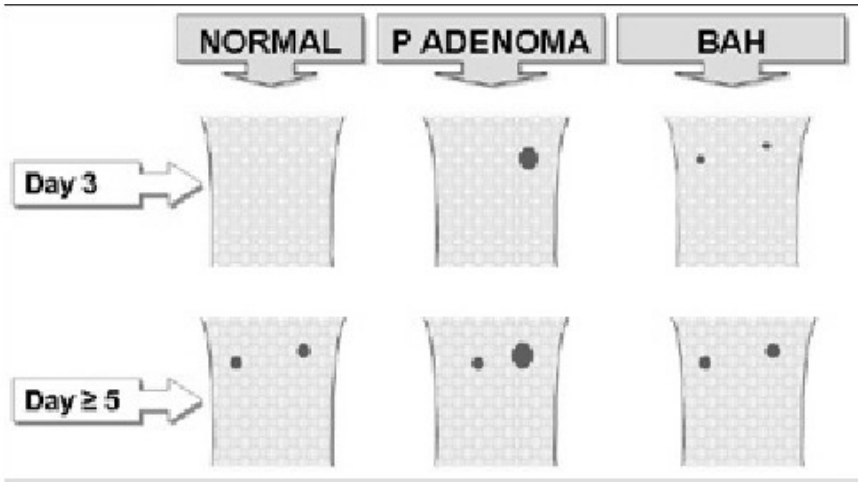
ADRENAL SCAN



TRACER	TIME (DAYS)	
	DEXAMETHASON SUPRESION	
	NO	YES
^{131}I-NP59	5 и/или 7	3-5 и 7
SCAN TIME	2-3	≥ 5

ADRENAL SCAN

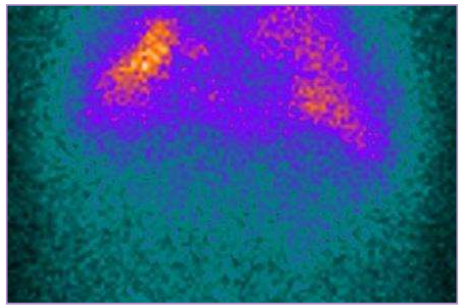
Dexamethasone suppresses ACTH and reduces TRACER binding in the zona fasciculata. This increases the sensitivity of the findings. It is important to discontinue all drugs that affect the HPA/RAA axis, in order not to disturb the scintigraphic display.



ADRENAL SCAN

- ^{131} I-6-β-iodomethyl-19-norcholesterol

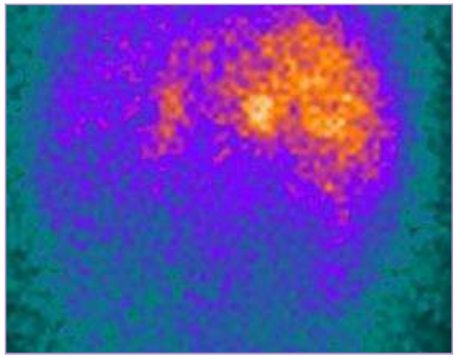
1 D



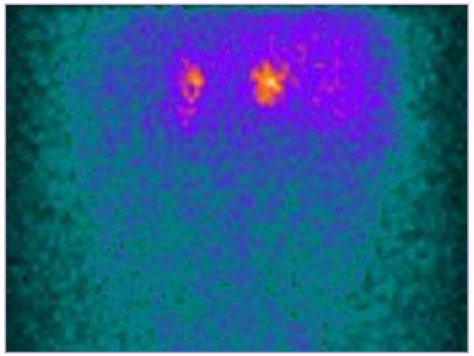
NO UPTAKE

PHYSIOLOGICAL UPTAKE

5 D



7 D



CUSHING'Syndrome

- Elevation of cortisol level , which occurs in one of four ways

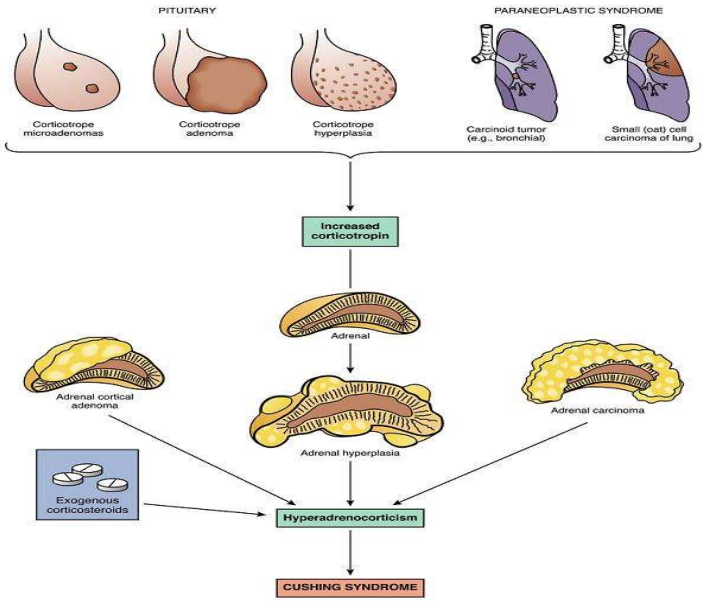
A- Endogenous causes :

i- ACTH*secreting pituitary microadenoma, few macroadenomas, OR hyperplasia (CUSHING's DISEASE)

ii-Adrenal tumor or hyperplasia

iii- Paraneoplastic syndrome

B- Exogenous cause :
Steroid Therapy



HYPERALDOSTERONISM :

- Excess level of aldosterone cause sodium retension, potassium excretion, resulting in hypertension & hypokalemia.

A- Primary : Conn Syndrome

- Caused by Adenoma (80%) F:M is 2:1
  Single or multiple
- Or primary adrenal hyperplasia (15%) ,
- Carcinoma is rare

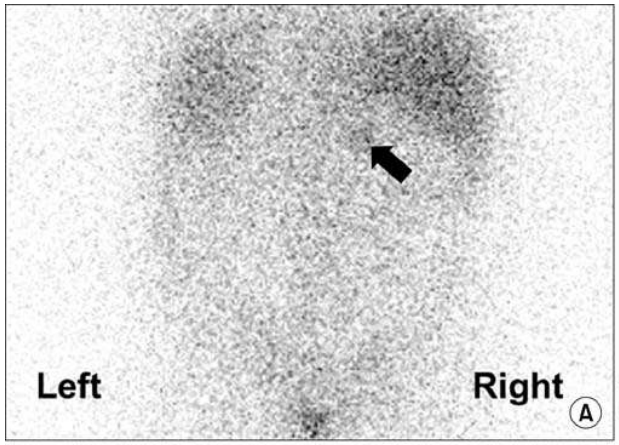
- Adjacent adrenal cortex is NOT atrophic
- There is ↑aldosteron→ Na retention & K excretion
  ↑ BP , Hypokalemia , ↓RENIN

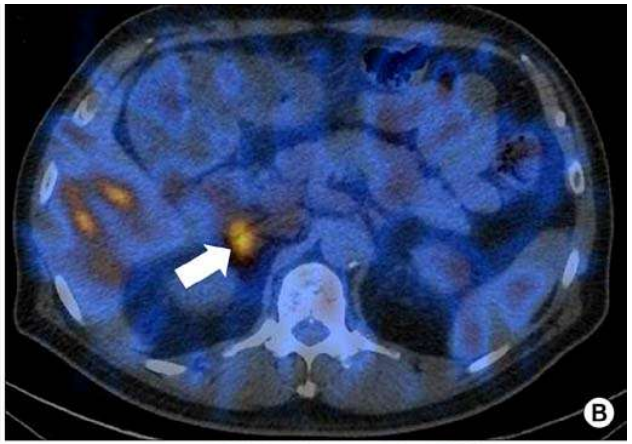
<u>Correctable cause of HYPERTENSION</u>

B- Secondary :

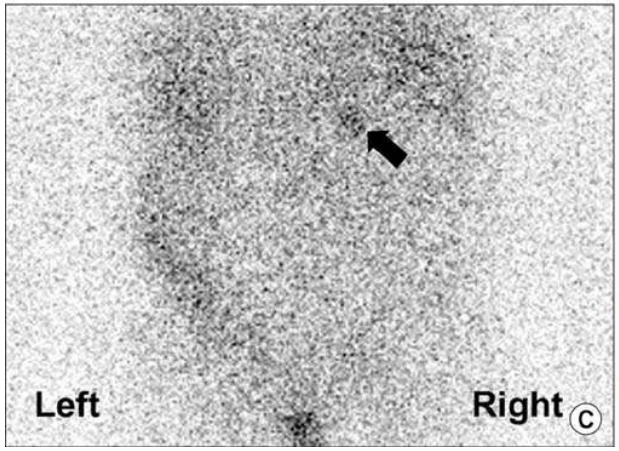
- Due to decreased renal perfusion,
- activation of the renin - angiotensin system → ↑aldosteron
- Differentiate from primary by ↑ RENIN

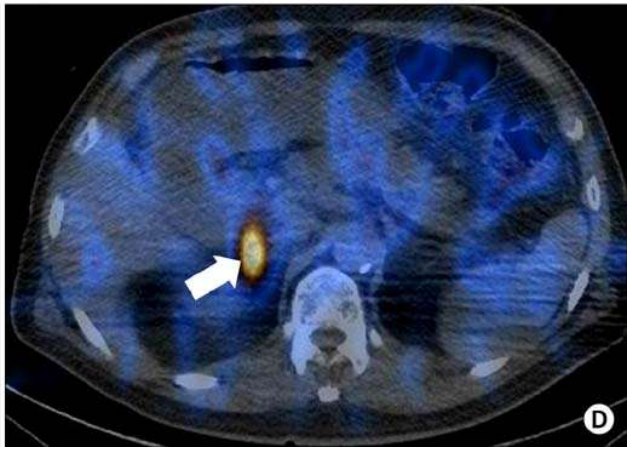
1 dan



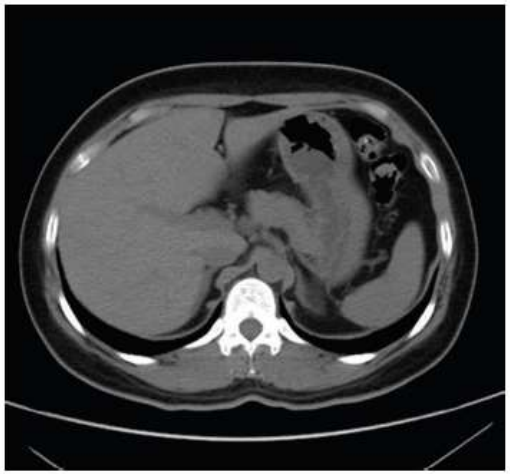


5 dan

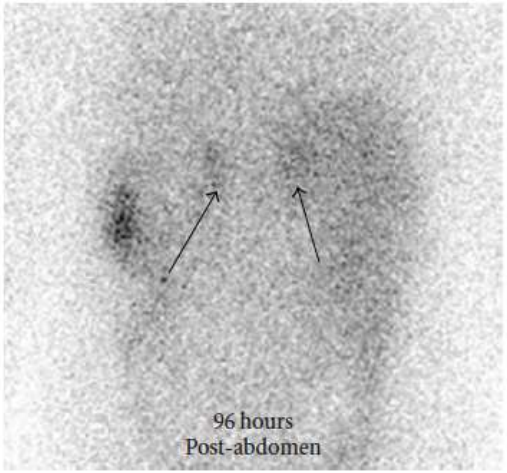




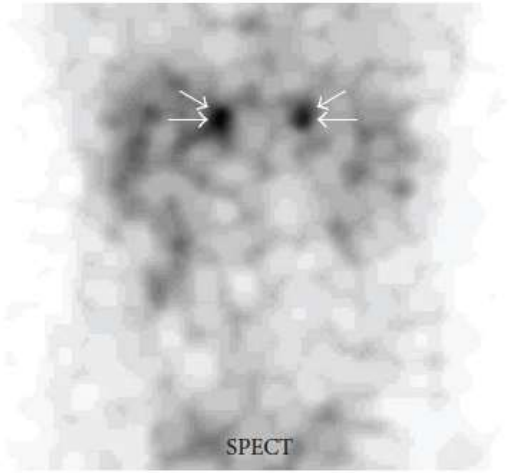
SPECT-CT -^{131} I-6-β-iodomethyl-19-norcholesterol
Adenoma gl. suprarenalis l. dex. (10mm)

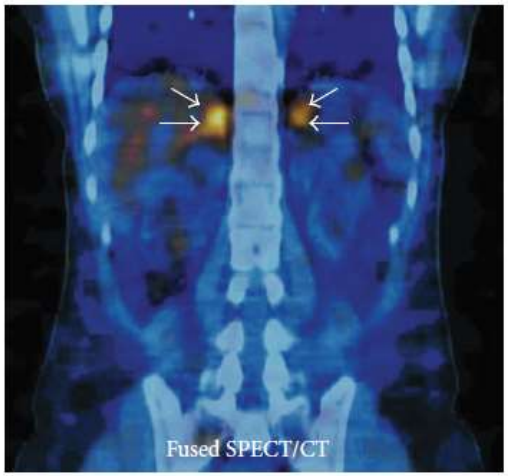


(a)



(b)

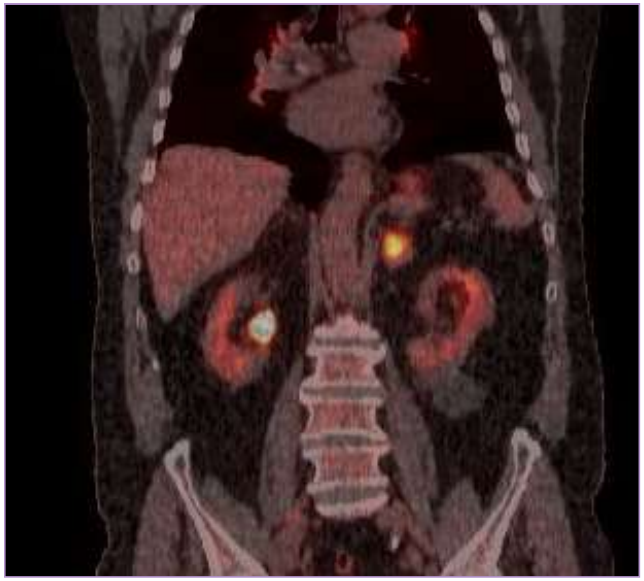


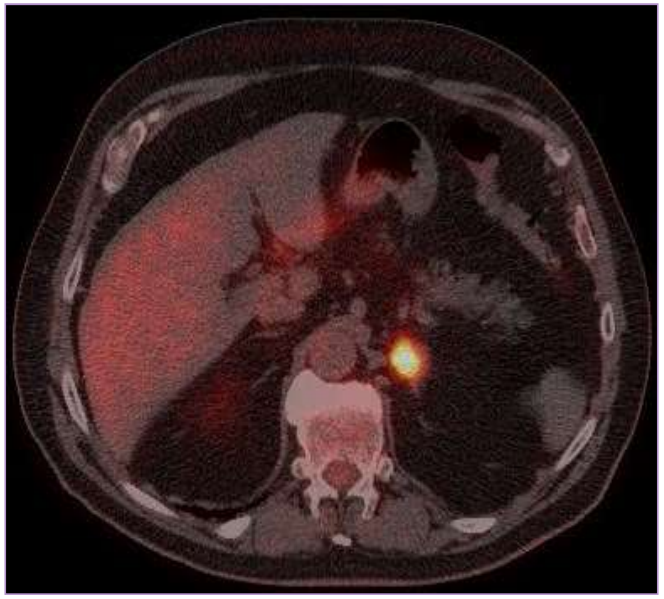


SPECT/CT ^{131}I- NP-59 BILLATERAL HYPERPLASIA

PET ^{18}FDG

CA gl. suprarenalis l. sin

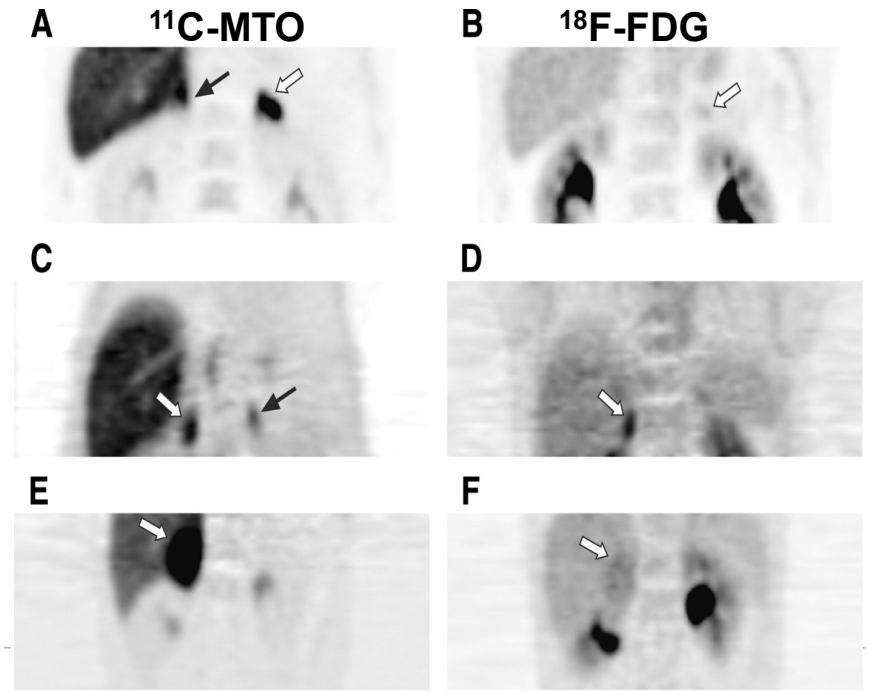




PET

^{11}C-metomidate (MTO), 3H- metirapon, ^{18}F-fluoroetiletomidat

- Binding for 11-βhidroksilaze (CYP 11B1, $P450_{11}$) cortisol and aldosteron enzimes



Adenoma l. sin,

Carcinoma l. dex.
Adenoma l.sin.

Adenoma l. dex.

THE ADRENAL MEDULLA :

- Composed of CHROMAFFIN CELLS & nerve endings
- Secretetes cholamines in response to sympathetic stimulation
- Also present in extra-adrenal sites (paragangliomas)

- Pathology includes tumors :

A- Pheochromocytoma

B- Neuroblastoma

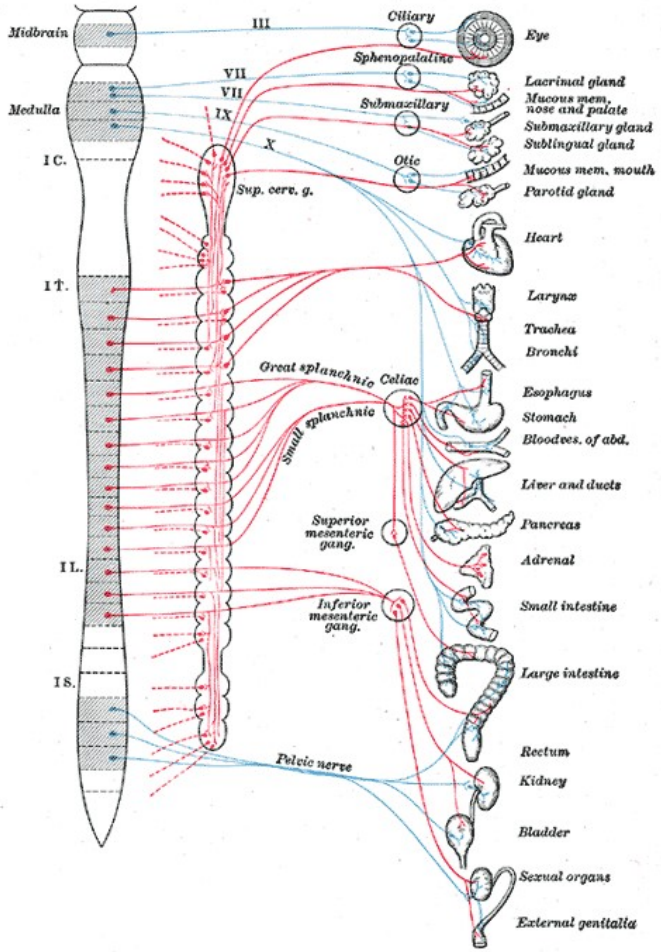
PHEOCHROMOCYTOMA :

- Secretes catecholamines → VMA
- Sometimes described as <u>The 10% Tumor</u> because :
    - * 10% bilateral
    - * 10% extra adrenal (Paraganglioma)
    - * 10% familial, maybe part of MEN syndrome
    - * 10% Malignant
- Usually well circumscribed,small to large in size,maybe pleomorphic. Malignancy confirmed by METASTASES
- Clinically sustained or paroxysmal attacks of ↑ BP
- CORRECTABLE cause of HYPERTENSION

ПАРАГАНГЛИОМИ – ектопично хромафино ткиво

- 87% абдомен
- 10% торакс
- 3% врат и глава
- 46% горњи парааортални г.
- 29% доњи парааортални г.
- 10% мокраћна бешика
- 3% мала карлица

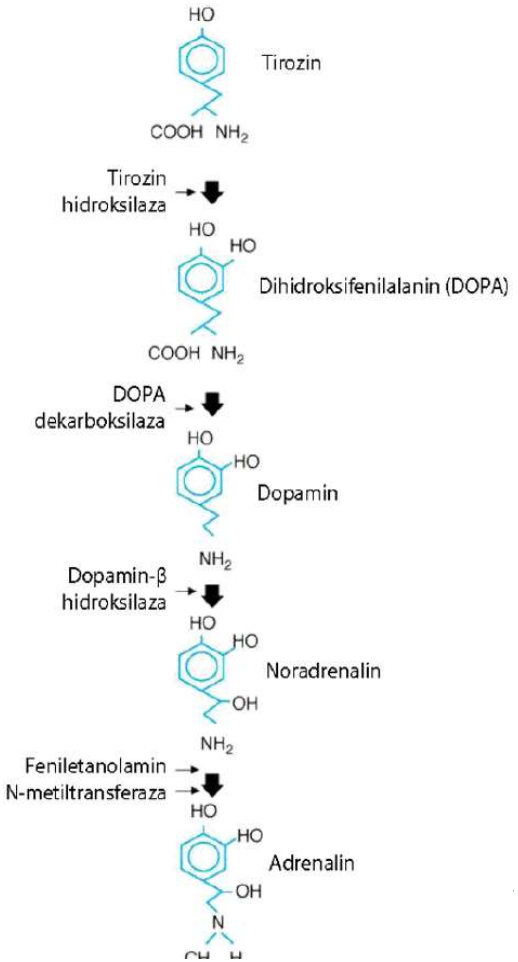
- НЕУРОБЛАСТОМ је најчешћи екстракранијални тумор код деце (2-4год). Представља тип НЕТ-а који настаје ћелија неуралног гребена које мигрирају дуж симпатикоадреналног система.
- 90% лучи допамин, АД, НАД и њихове метаболите

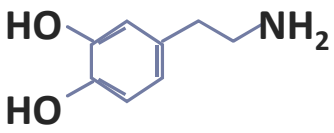


NEUROBLASTOMA :

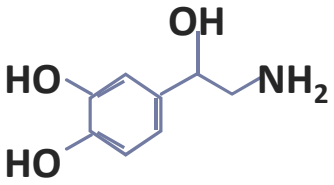
- Commonest extracranial solid tumor of childhood
- Usually adrenal but maybe extra-adrenal
- Familial or sporadic
- Associated with deletion of short arm of chromosome 1
- 90% associated with catecholamine secretion
- VMA excreted in 24 hr. urine helpful in diagnosis.
- Morphologically it is composed of small round blue cells which may differentiate to ganglion cells
- Spread to adjacent organs, lymph nodes, renal vein.
- Prognosis : STAGE , AGE , N myc amplification

ADRENAL MEDULLA SCAN

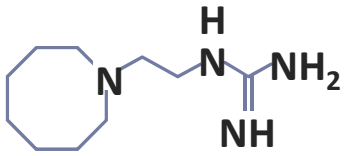




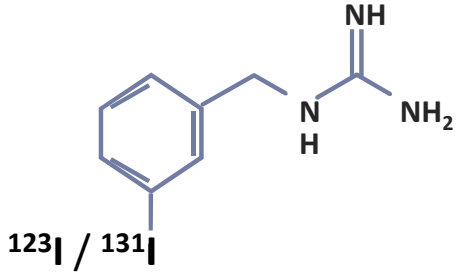
Adrenaline



Noradrenaline

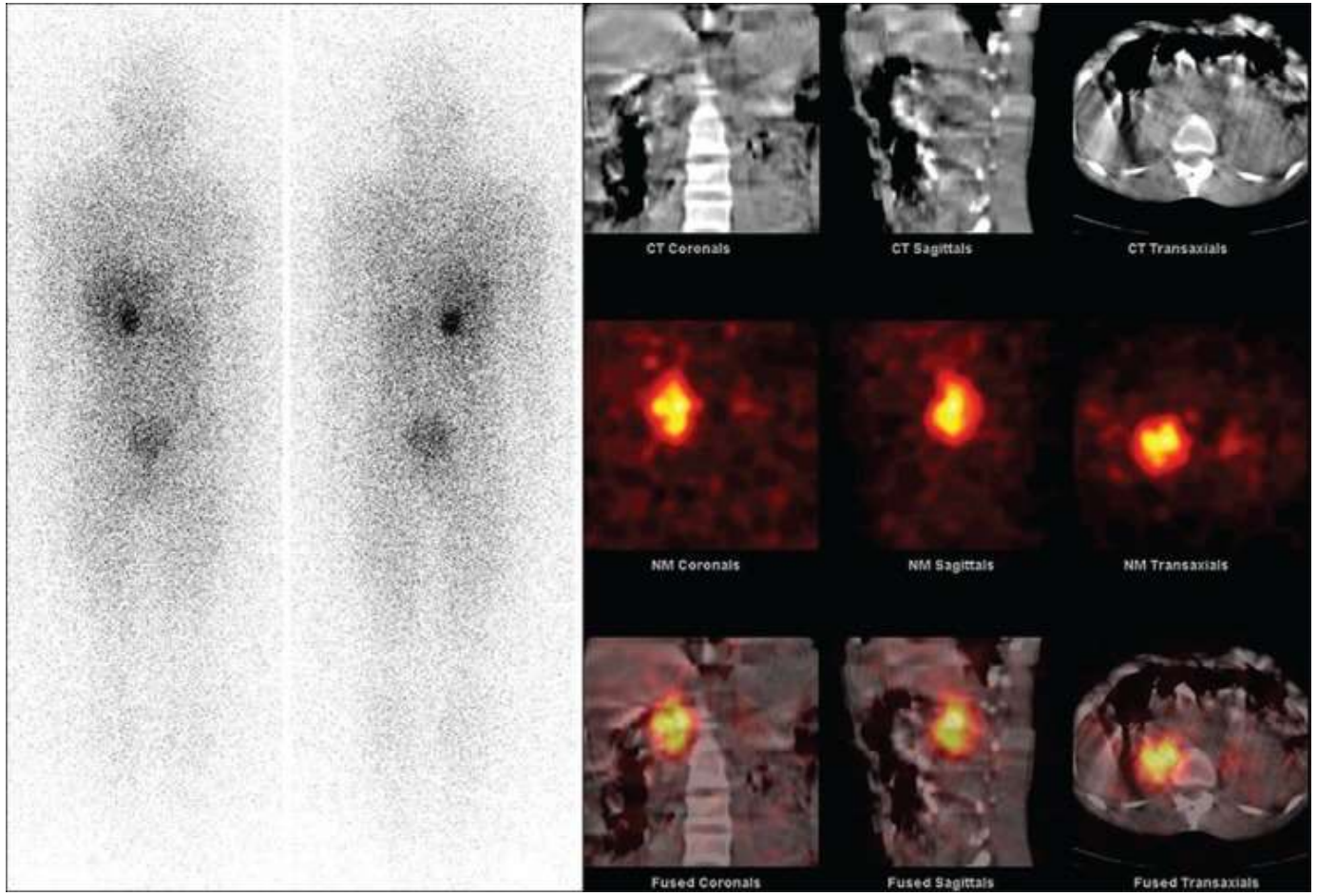


Guanethidine

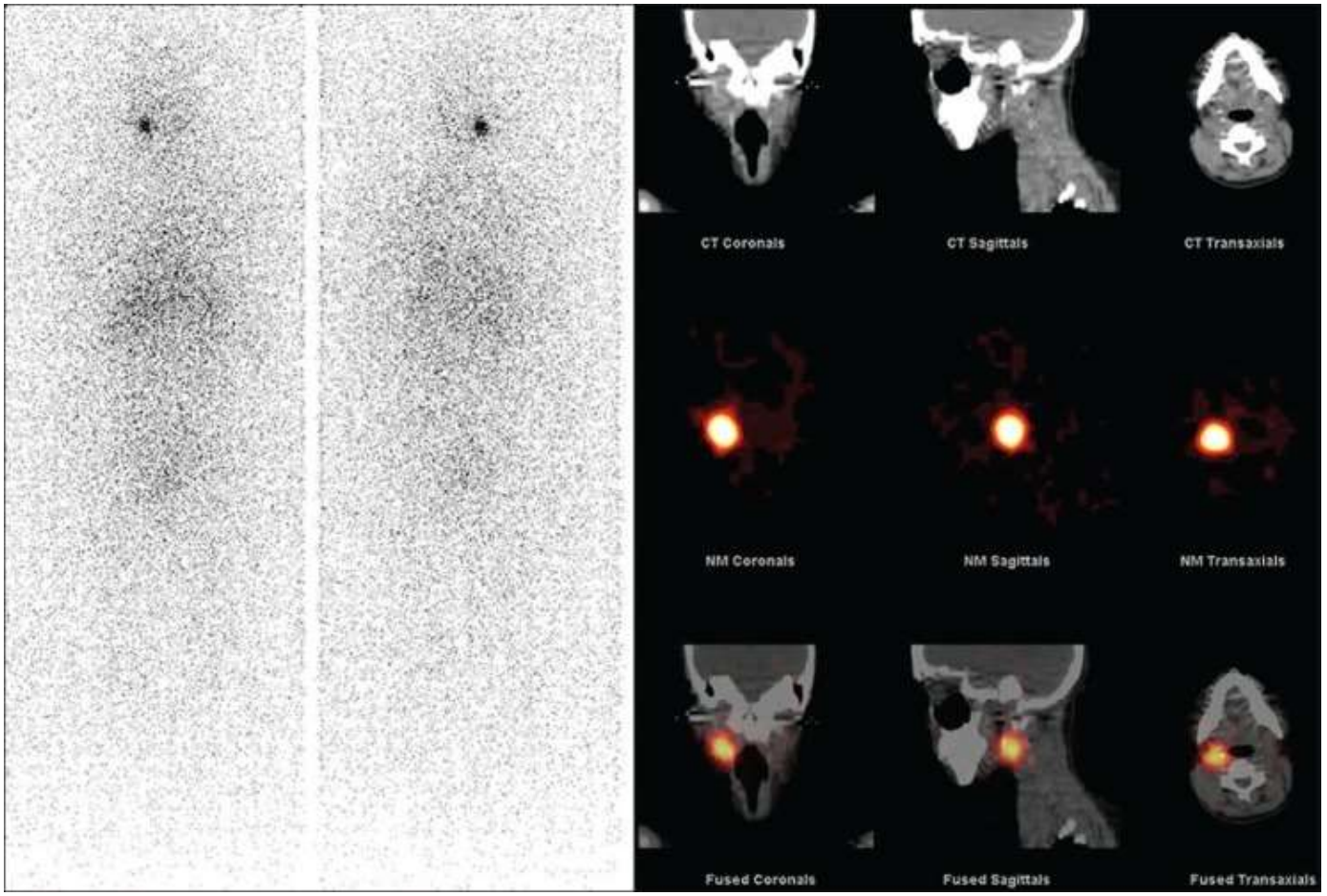


Meta-iodo-benzylguanidine (MIBG)

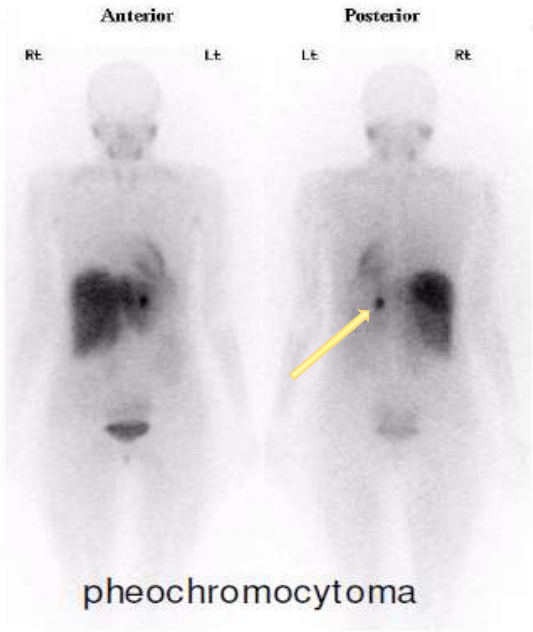
- the structure corresponds to noradrenaline (NA), it is actively taken up on the membranes of symp. n.s. and adrenal medulla, does not bind to postsynaptic receptors and is not metabolized it is transported and retained for a prolonged period in catecholamine storage vesicles, which is the basis for its highly specific accumulation and recording



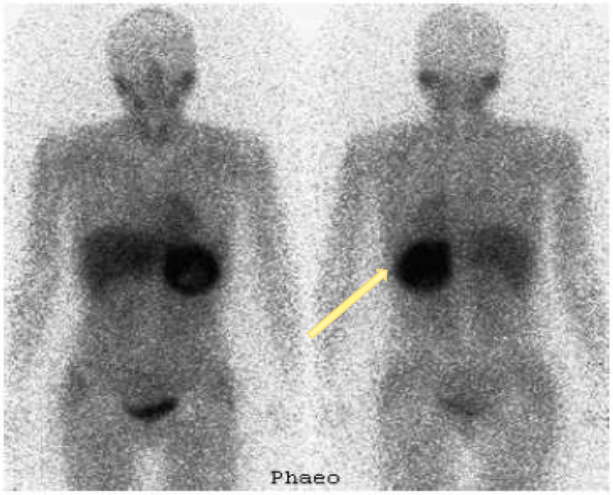
^{131}I-mIBG Pheochromocytoma gl. suprarenalis dex.

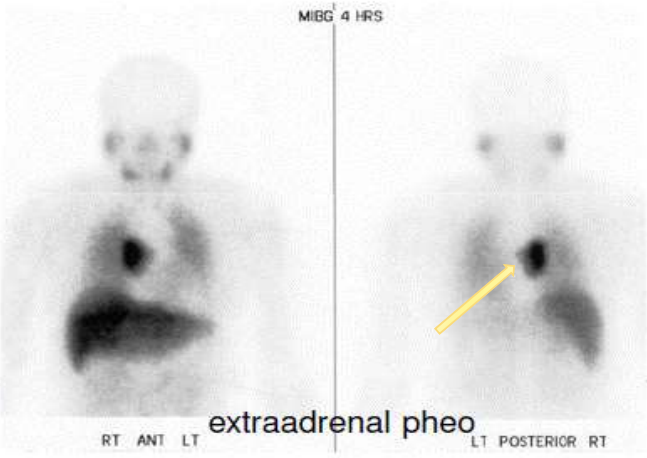


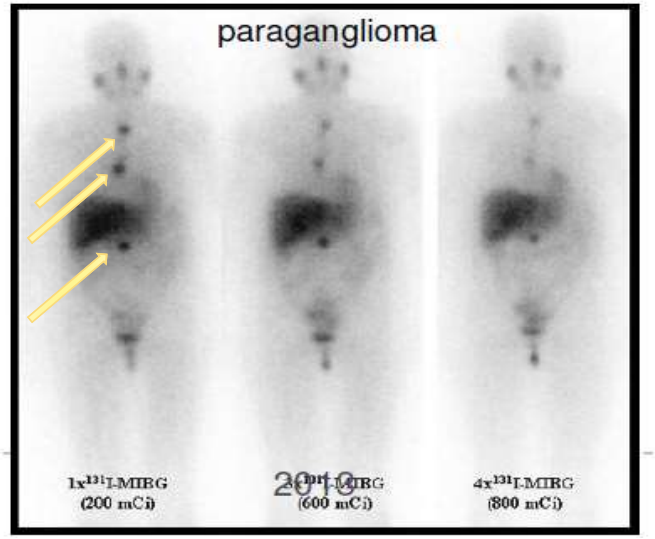
^{131}I-mIBG - Paraganglioma carotid body dex.


Anterior
Posterior
Rt
Lt
Lt
Rt
pheochromocytoma

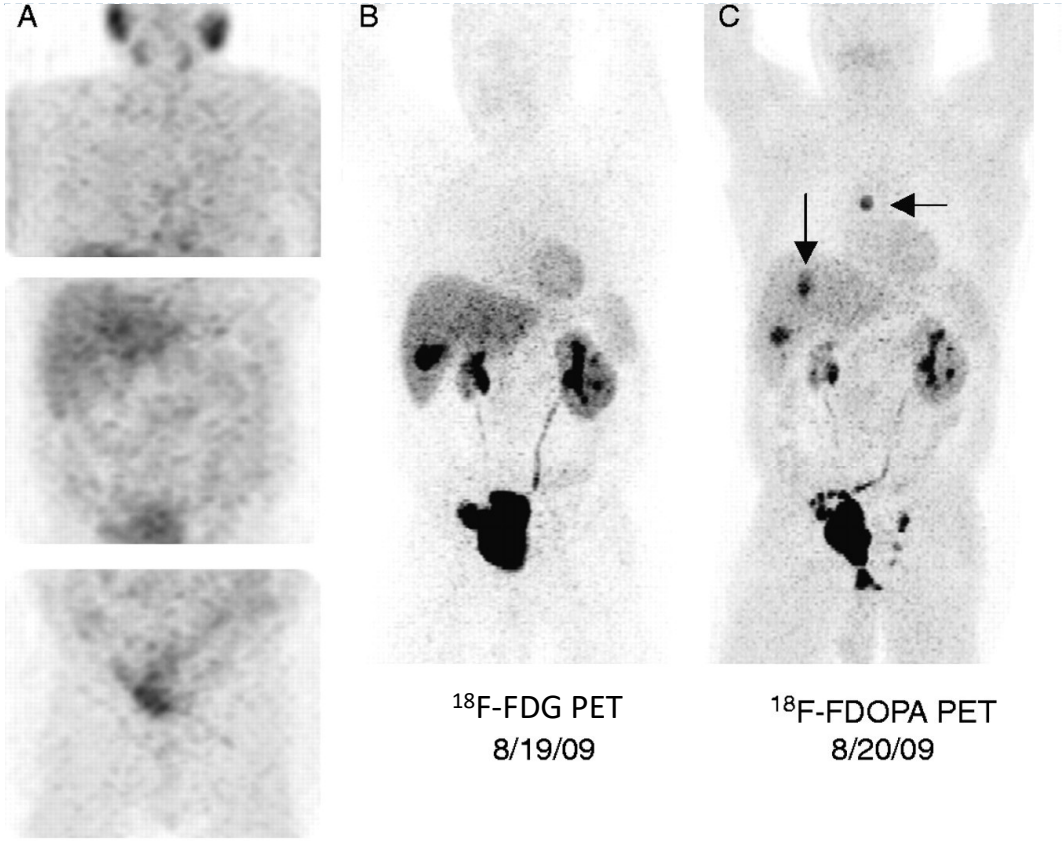
^{123}I-MIBG


Phaeo


MIBG 4 HRS
RT ANT LT
LT POSTERIOR RT
extraadrenal pheo

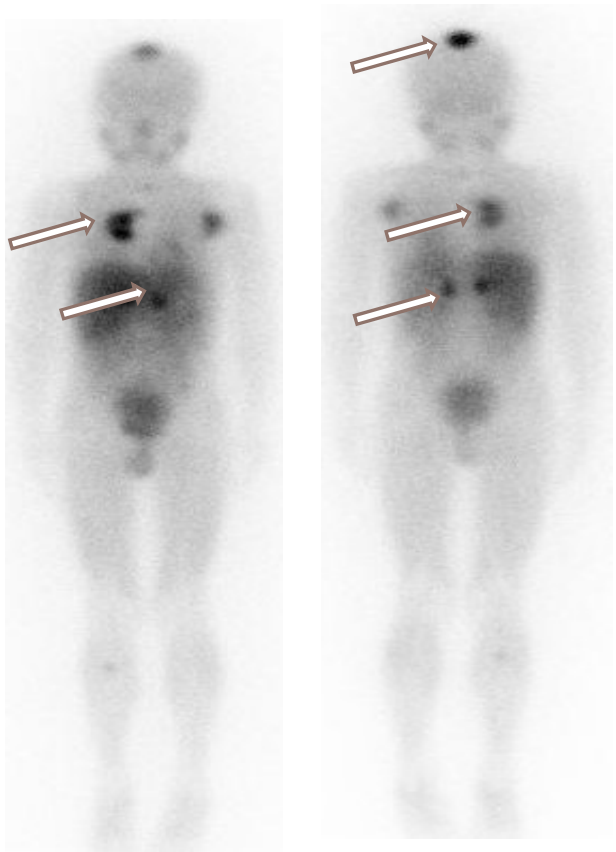

paraganglioma
1x131I-MIBG
(200 mCi)
2x131I-MIBG
(600 mCi)
4x131I-MIBG
(800 mCi)

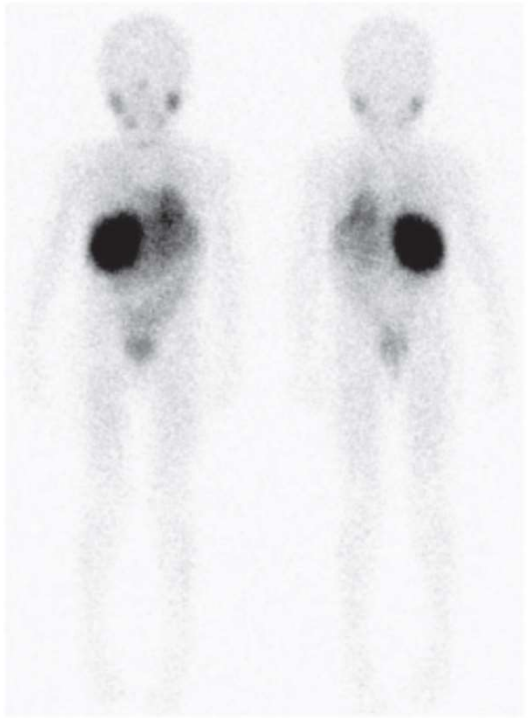
PET - paraganglioma

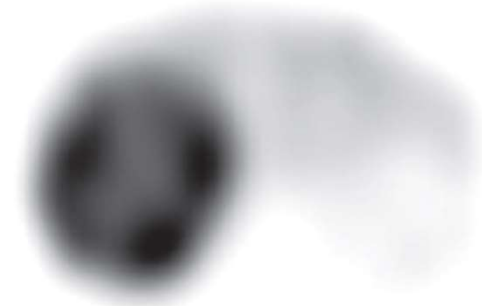


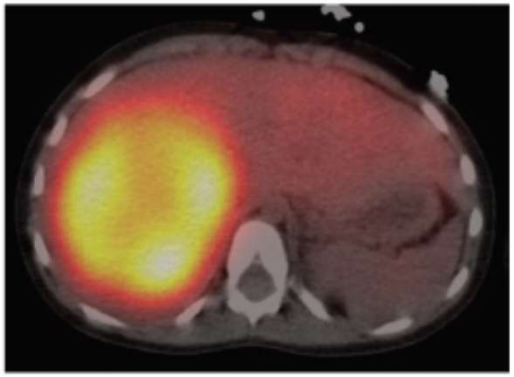
NEUROBLASTOMA

^{131}I-mIBG

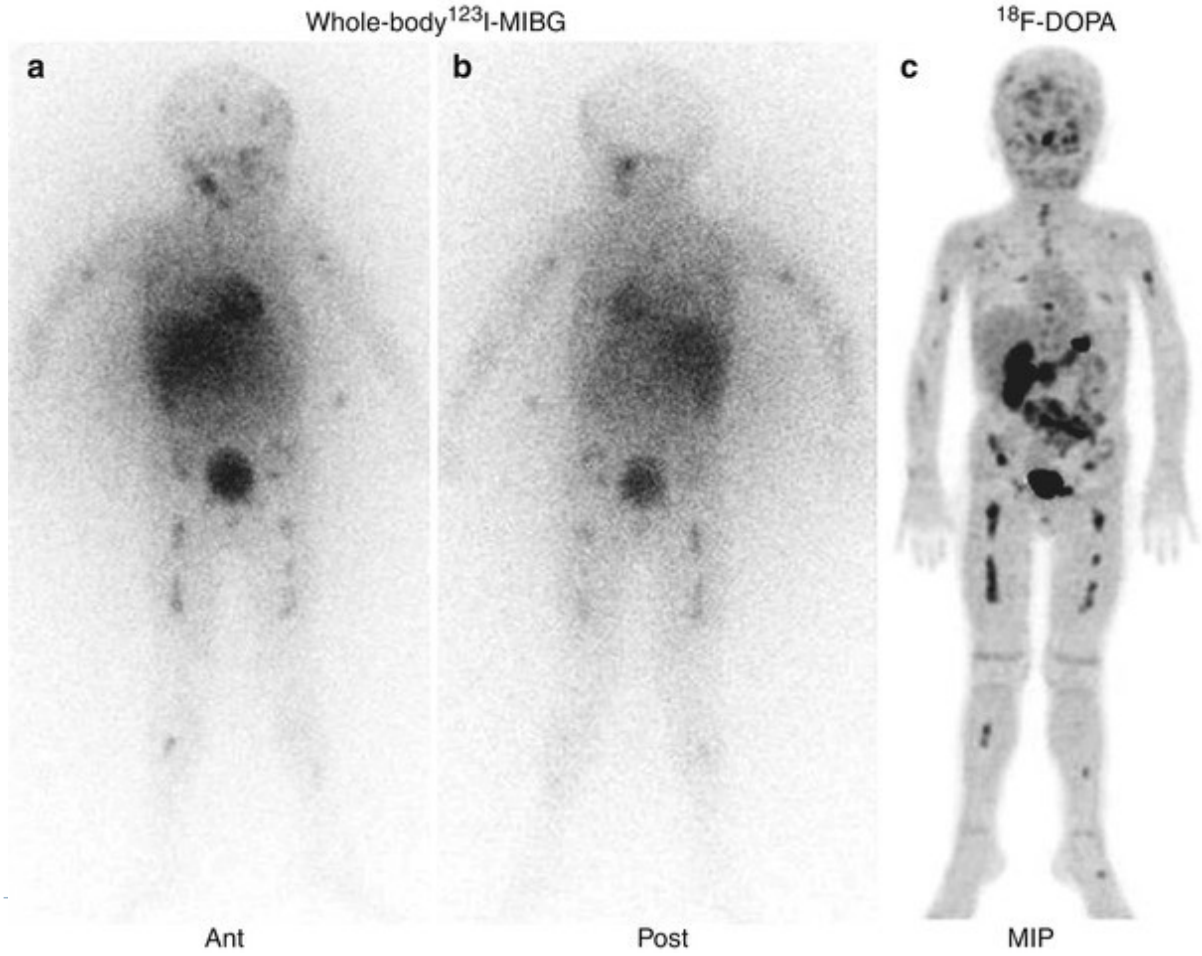






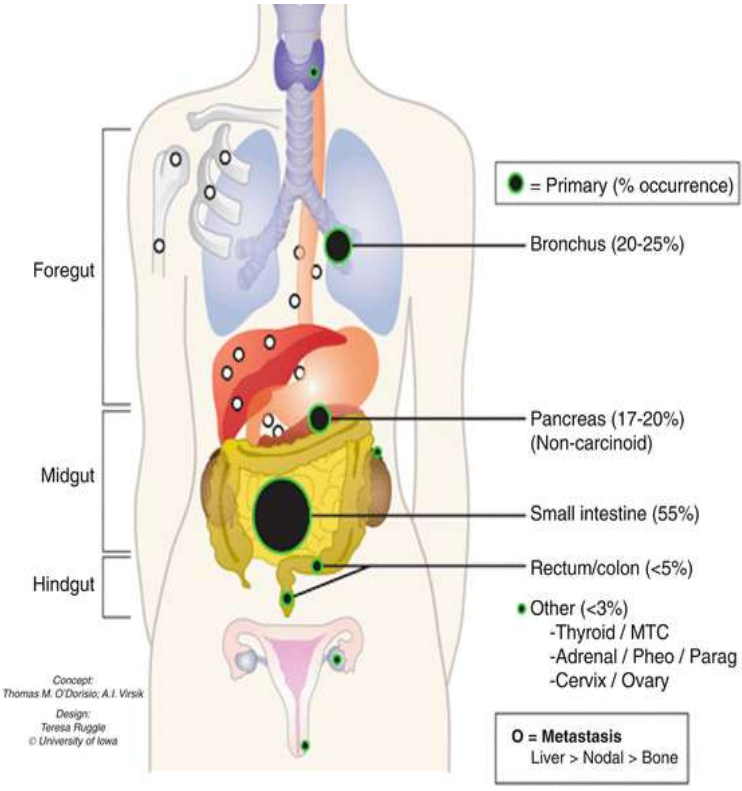


NEUROBLASTOMA



GASTROENTEROPANCREATIC NEUROENDOCRINE TUMORS (GEP-NETS

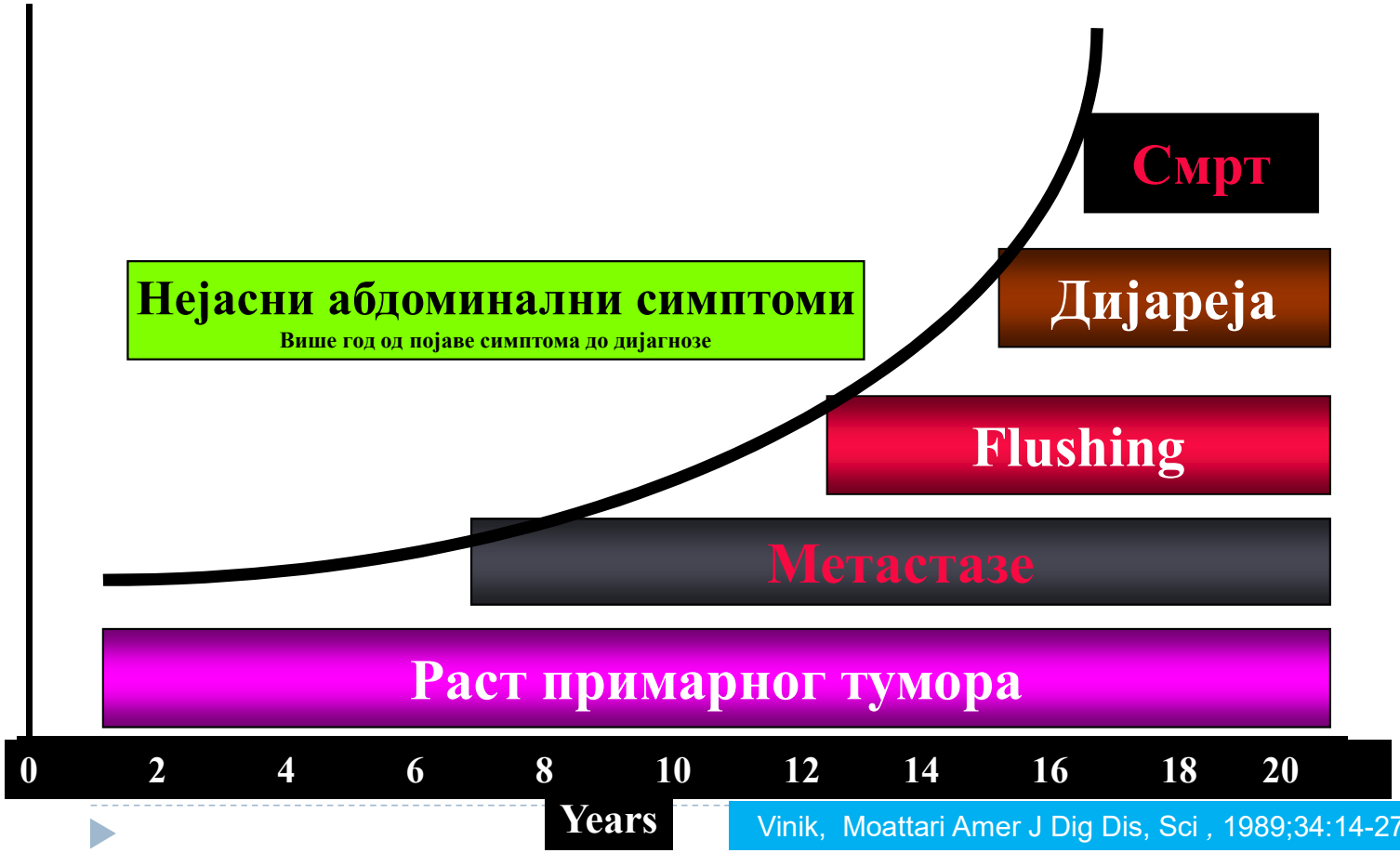
- also known as carcinoids and islet cell tumors, are tumors derived from neuroendocrine cells that can occur anywhere along the gastrointestinal tract and comprise a heterogeneous family of neoplasms with a wide and complex spectrum of clinical behavior.
- Serbia incidence - 100/god



Grade	G1	G2	G3
Ki67 index (%)**	≤2	3–20	>20
MI (mitotic count)*	<2	2-20	>20

Clinical syndrome	Tumor site	clinical behavior
Carcinoid syndrome	Pulmo, Gastroduodenum and Pancreas = Foregut	Serotonin, Histamin, Tahikini, Bradikinin
	Ileum and Jejunum = Midgut	Serotonin, Tahikinini, Bradikinin
Zollinger Ellison syndrome	Pancreas ,Duodenum	Gastrin
Hypoglycemic (Insulinoma)	Pancreas	Insulin, proinsulin IGF-I/II
Verner-Morrison (WDHA) syndrome	Pancreas, Ganglioneuromas, Paraganglioma, Pulmo	VIP
Glucagonoma syndrome	Pancreas Duodenum	Glukagon
Somatostatinoma syndrome	Pancreas, Colon	Somatostatin, CgA, HCG-α/β, PP
“Non-functioning” Tumours		

DESEASE PROGRESSION



- Црвенило-зајапуреност
  - Без знојења
  - Невезано за алкохол
- Дијареја
  - Нарочито ноћу
- Иритабилни колон
- Надимање
- Гасови

Vinik, Moattari Amer J Dig Dis, Sci , 1989;34:14-27.

Dg management

	Well-differentiated		Poorly differentiated
Grade (ENETS)	Low (G1)	Intermediate (G2)	High (G3)
Ki-67 index (%)	≤2	3-20	>20
Anatomic imaging	more rapid growth on serial imaging		
Functional imaging	Octreoscan SPECT or SSTR PET +ve → FDG PET +ve		
Prognosis	Indolent (slowly growing)		Aggressive
Treatment options	Surgery for localised +/- resectable metastatic disease		
	Observation Somatostatin analogues Radionuclide therapy		Chemotherapy
	Everolimus, sunitnib, α-interferon Liver metastases: radiofrequency ablation, hepatic embolisation, TACE, SIR-Spheres		

2016 ENETS Consensus Guidelines for the Diagnosis and Treatment of Neuroendocrine Tumors

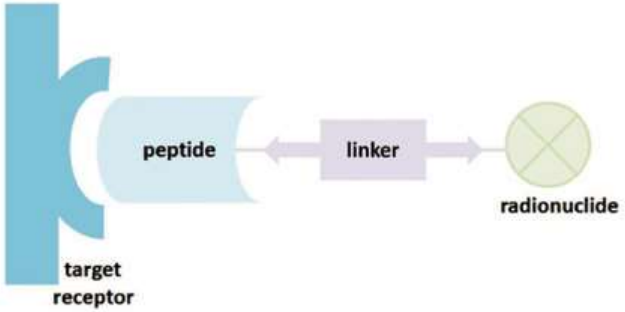
SOMATOSTATIN RECEPTOR (SSTR) IMAGING

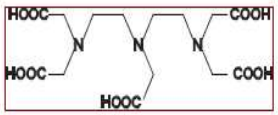
Somatostatin receptors (SSTR) are integral membrane glycoproteins of normal cells of neuroendocrine origin (islets of Langerhans of the pancreas, anterior lobe of the pituitary gland and C-cell of the thyroid gland), but also of well-differentiated brain tumors, malignant lymphomas, breast and lung and activated lymphocytes

The uptake of radiolabeled agonist or antagonist depends on:

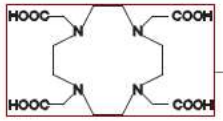
- ✓ Tissue blood supply
- ✓ Affinity to the receptor
- ✓ Stability of radiopharmaceuticals
- ✓ Concentration of receptors
- ✓ The presence of radiopharmaceutical competitors (physiological or pharmacological agonist/antagonist)

SOMATOSTATIN RECEPTOR IMAGING





DTPA
Dietilentriaminopentaacetat



DOTA
1,4,7,10 -tetraazaciklododekan-N,N,N', N''-tetraacetat

Tumor	SST1 (%)	SST2 (%)	SST3 (%)	SST4 (%)	SST5 (%)
Gastrinoma	79[a]	93	36	61	93
Insulinoma	76	81	38	58	57
Non-functioning pancreatic tumor	58	88	42	48	50
Carcinoid tumor of the gut	76	80	43	68	77

^{111}In-pentetreotid (Octreoscan®)

diethylenetriaminepentaacetic acid-d-phenylalanyl^{1}-octreotide

^{99m}Tc-HYNIC-TOC (Tektrotyd®)

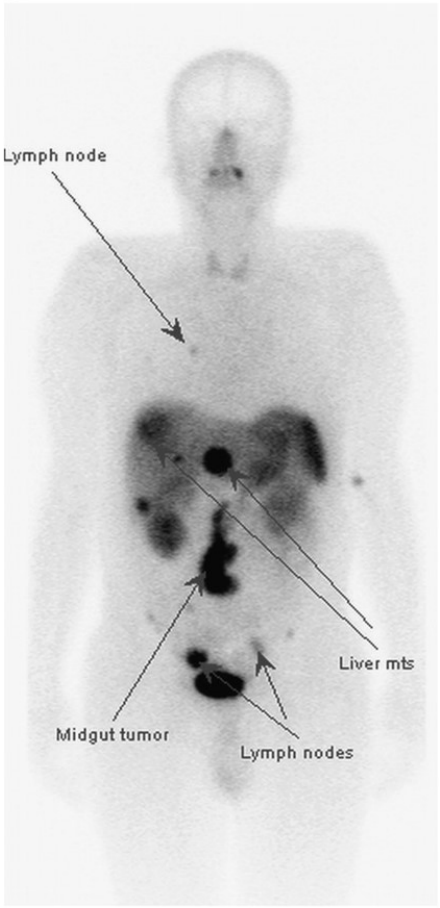
hydrazinonicotinyl acid-dphenylalanyl^{1}-tyrosine^{3}-octreotide

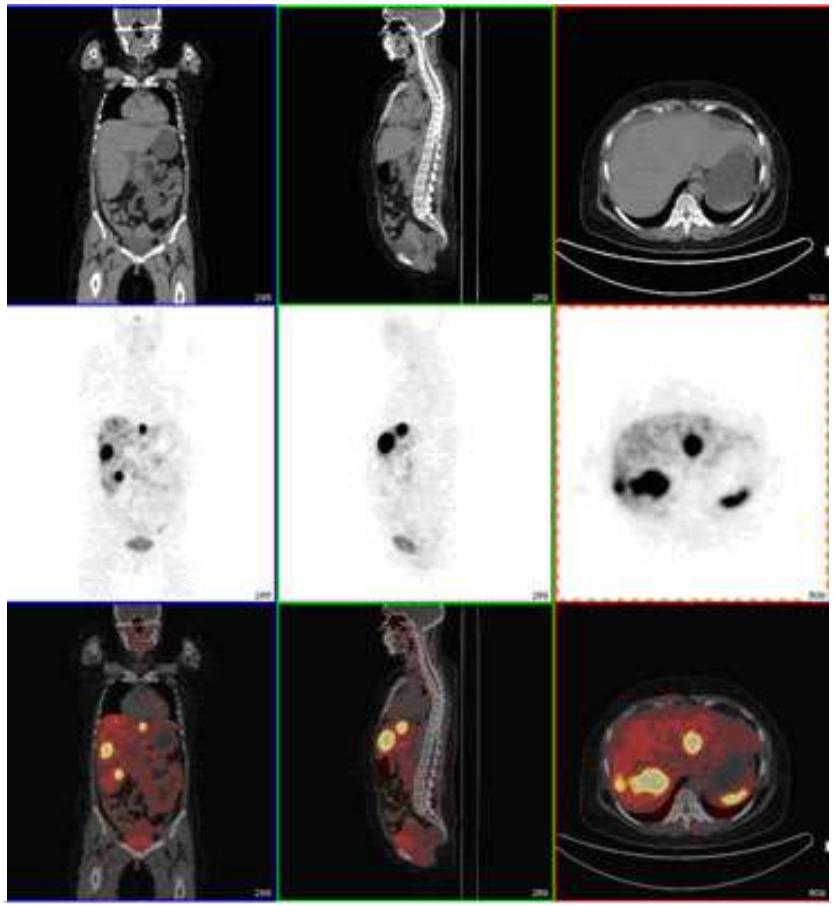
SOMATOSTATIN RECEPTOR IMAGING

Radioligand	Availability	Receptor binding data
^{123}I-octreotide	No longer in use	Not studied
^{123}I-Tyr^{3}-octreotide	No longer in use	hSSTR2, 5 (3)
^{111}In-DTPA-D-Phe^{1}-octreotide	Commercially available	hSSTR2, 5 (3)
^{111}In-DOTA-Tyr^{3}-octreotide	May be prepared in house	hSST2, 5 (3)
^{111}In-DOTA-lanreotide (MAURITIUS)	In house	hSST2–5 (1)
^{99m}Tc-HYNIC-octreotide	In house	Not studied
^{99m}Tc-depreotide (P829)	Commercially available	hSST2, 3, 5
^{99m}Tc-Demotate1 (Tyr^{3}-Octreotate analog)	May be prepared in house	Not studied
^{123}I-VIP	In house	hSSTR3, VIPR1, 2
^{99m}Tc-TP 3654 (VIP analog)	In house	not studied
^{99m}Tc-Neurotensin-XI	First clinical data	NTR1
^{111}In-DTPA-D-Glu^{1}-minigastrin	Phase I/II	CCK2
^{99m}Tc-RP527 (Bombesin/GRP analog)	First clinical experiences	GRP (BB2)
^{99m}Tc-Bombesin (BN)	First clinical experiences	Not studied
^{111}In-DTPA-Pro1, Tyr4-BN	In house	Not studied
PET-TRACER		
^{64}Cu-DOTA-Aoc-BN(7-14)	In house	Not studied
^{64}Cu-TETA-octreotide	In house	Not studied
^{68}Ga-DOTA-Tyr^{3}-octreotide	In house	hSSTR 2 (3,4,5)
^{68}Ga-DOTA-Tyr^{3}-octreotate	In house	
^{18}F-FP-Gluc-TOCT (octreotate analog)	In house	hSSTR2 (3,4)

SOMATOSTATIN RECEPTOR IMAGING

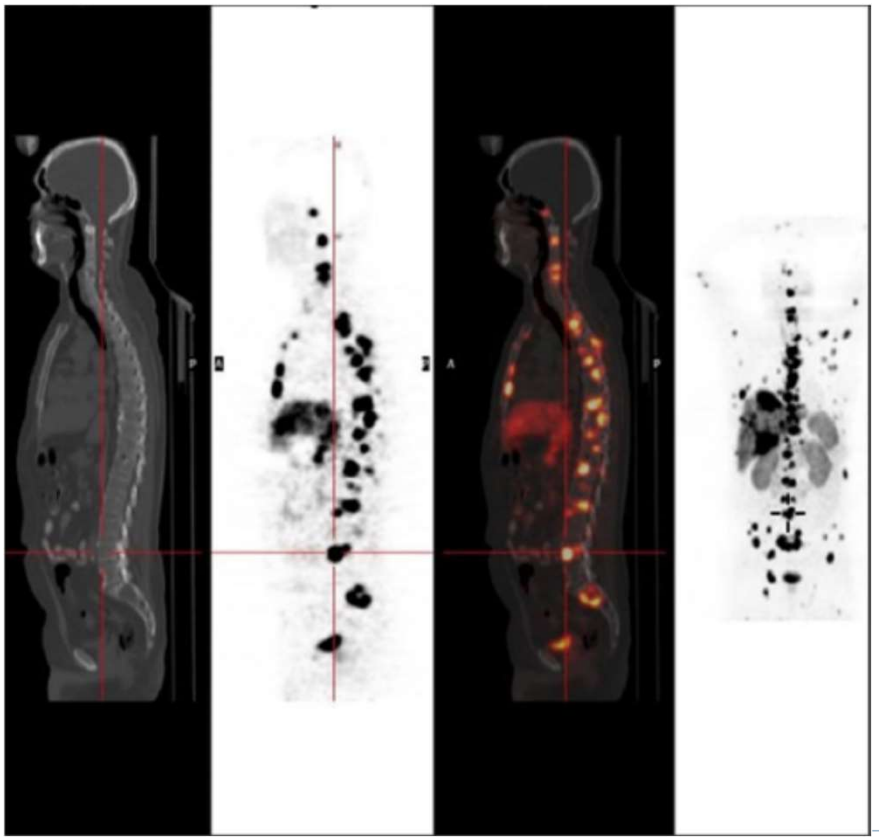
^{99m}Tc-Tektrotyd
(tyr^3-octreotyd)

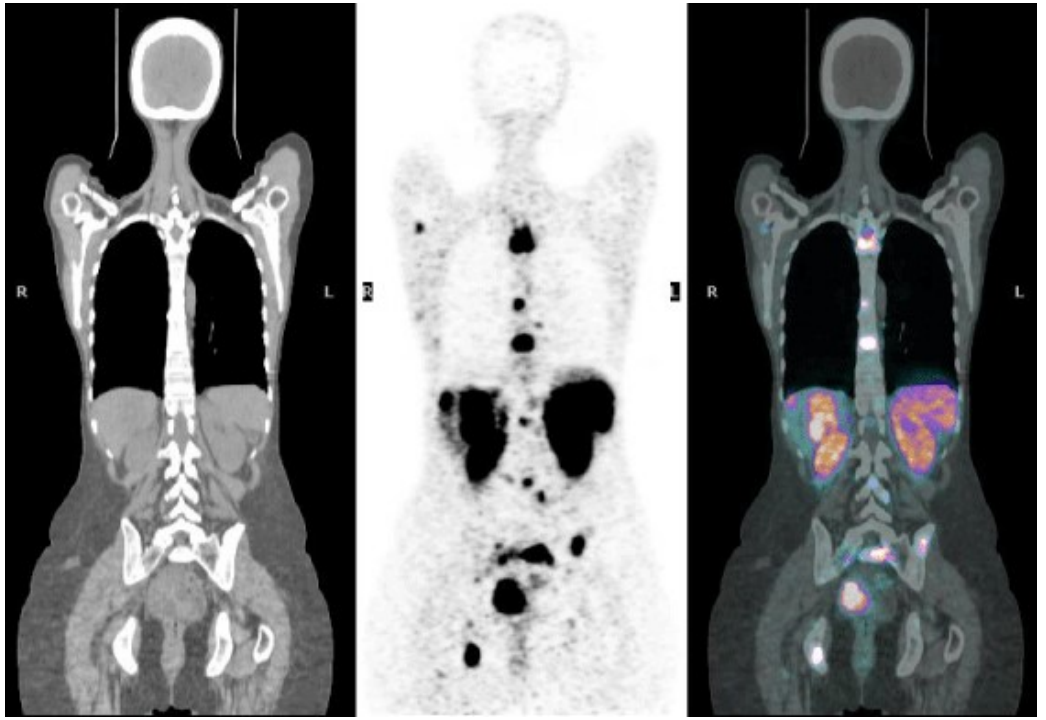




SOMATOSTATIN RECEPTOR IMAGING

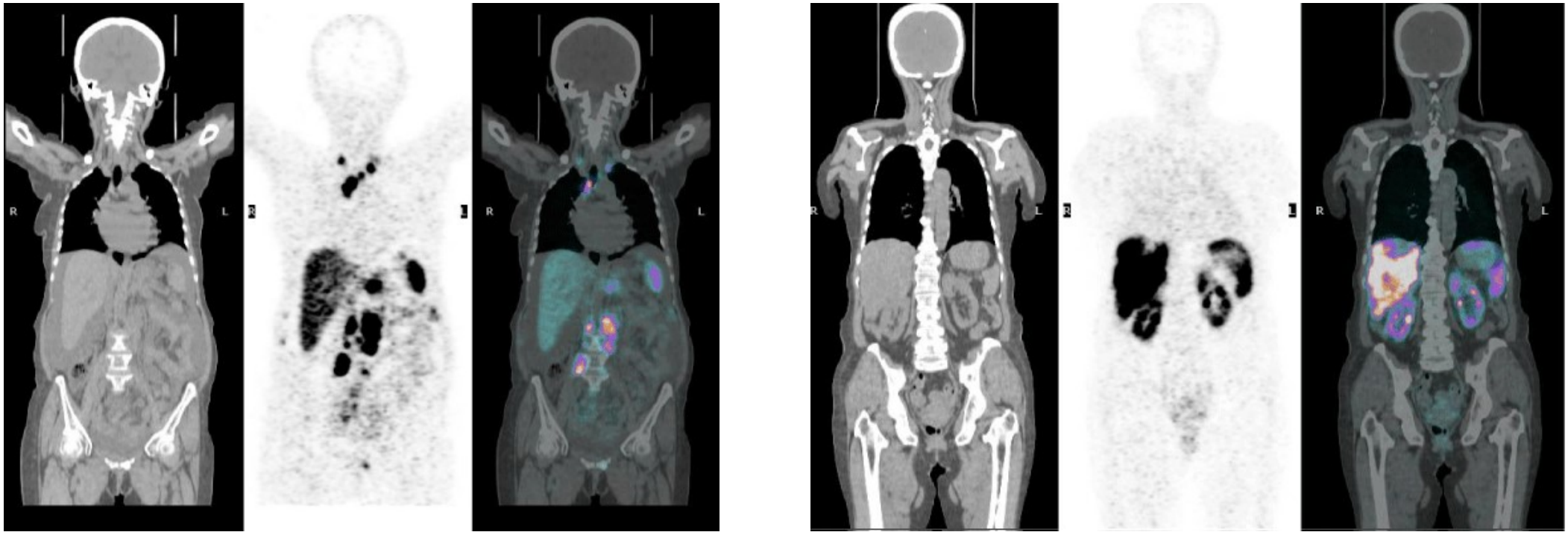
^{68}Ga-DOTATOC



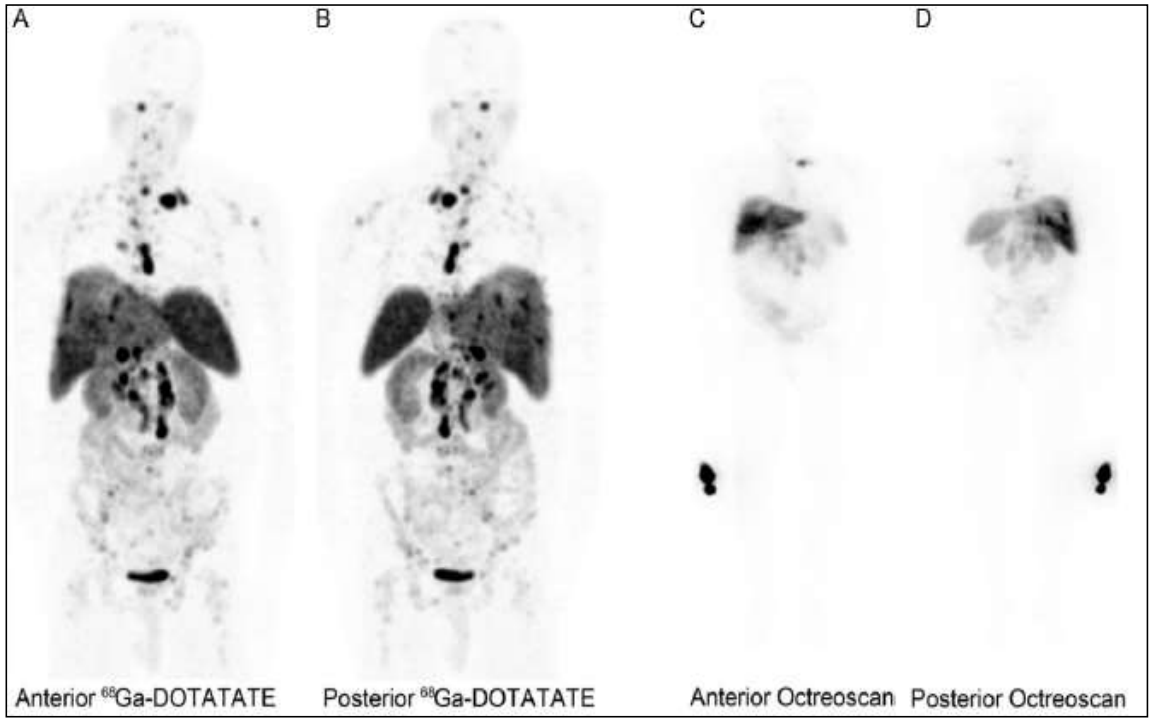


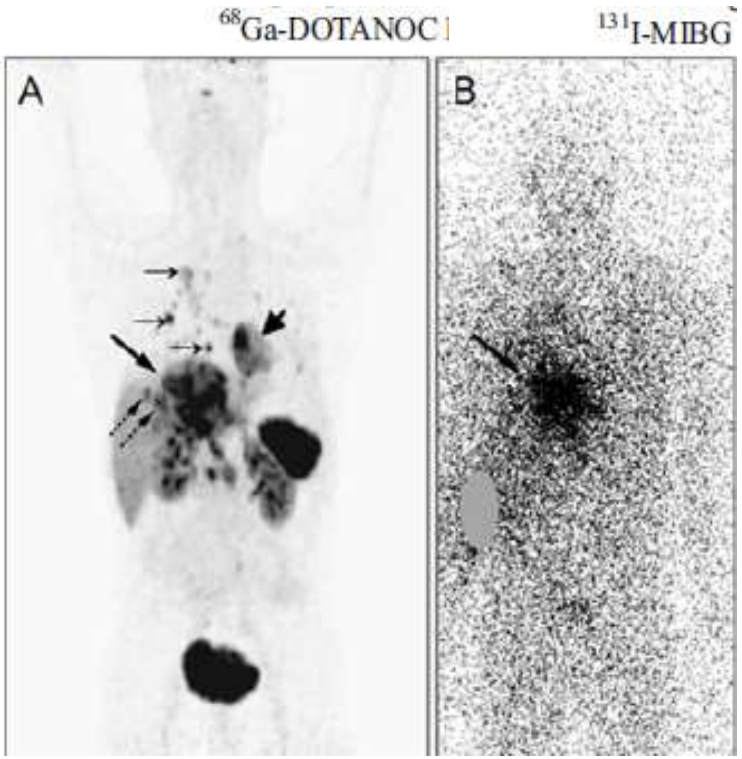
SOMATOSTATIN RECEPTOR IMAGING

^{68}Ga-DOTATATE

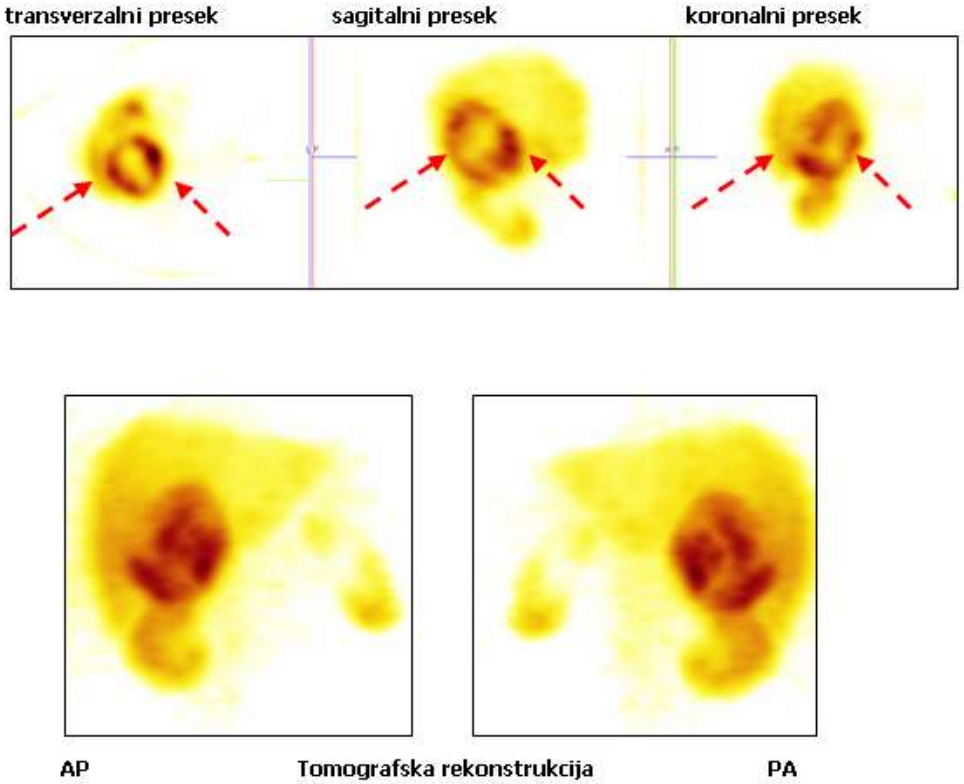


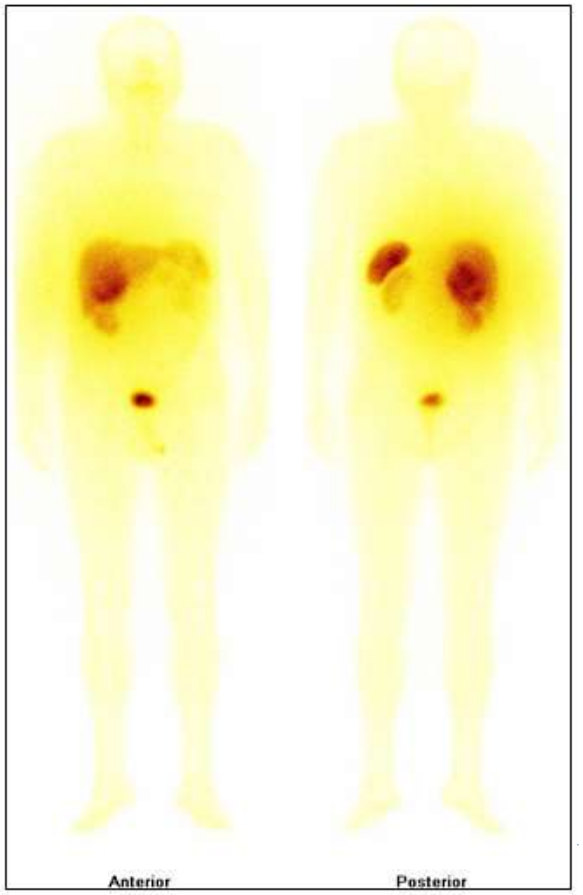
SOMATOSTATIN RECEPTOR IMAGING





SOMATOSTATIN RECEPTOR IMAGING



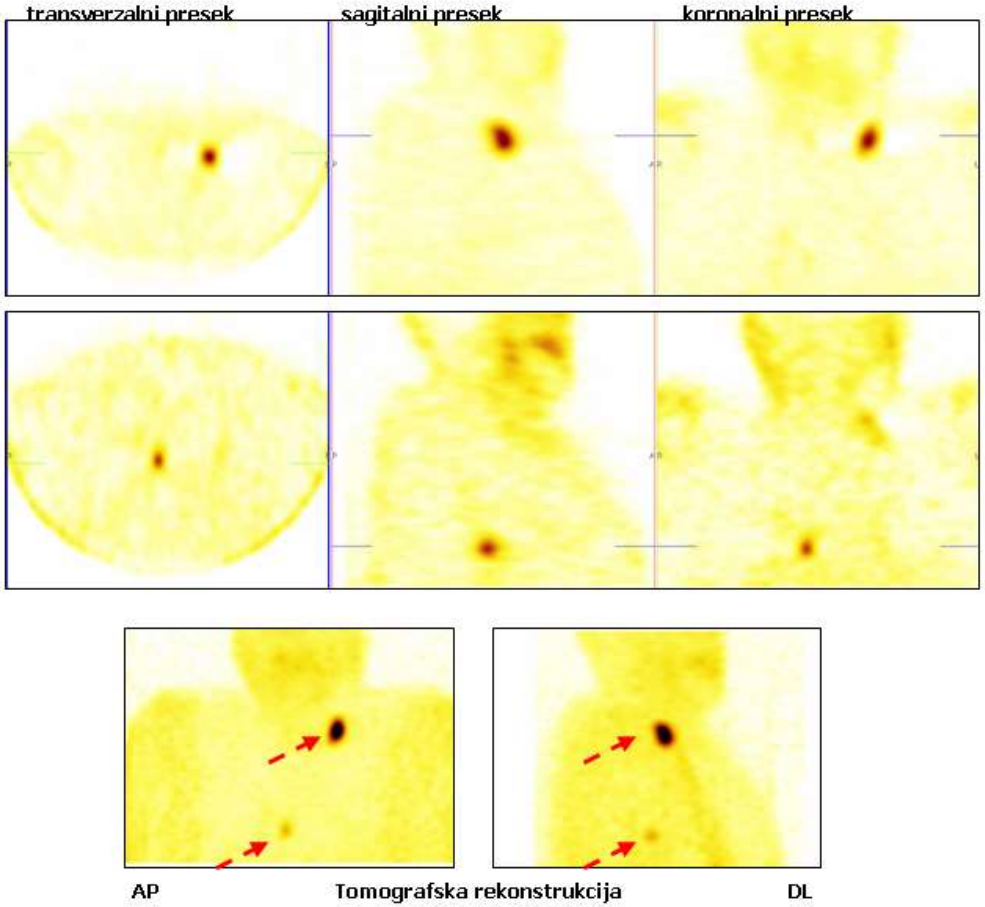


^{99m}Tc-Tektrotyd (tyr^{3}-octreotyd)

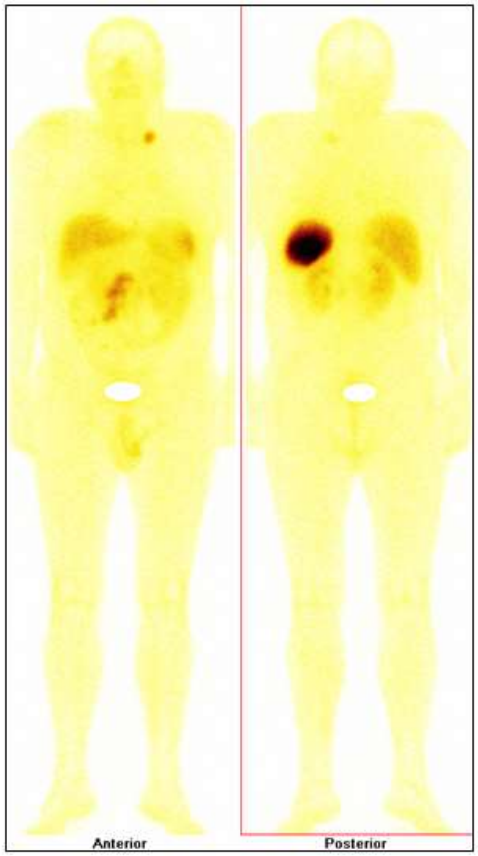
Pheochromocytoma gl. suprarenalis dex

SOMATOSTATIN RECEPTOR IMAGING

^{99m}Tc-Tektrotyd (tyr^3-octreotyd)

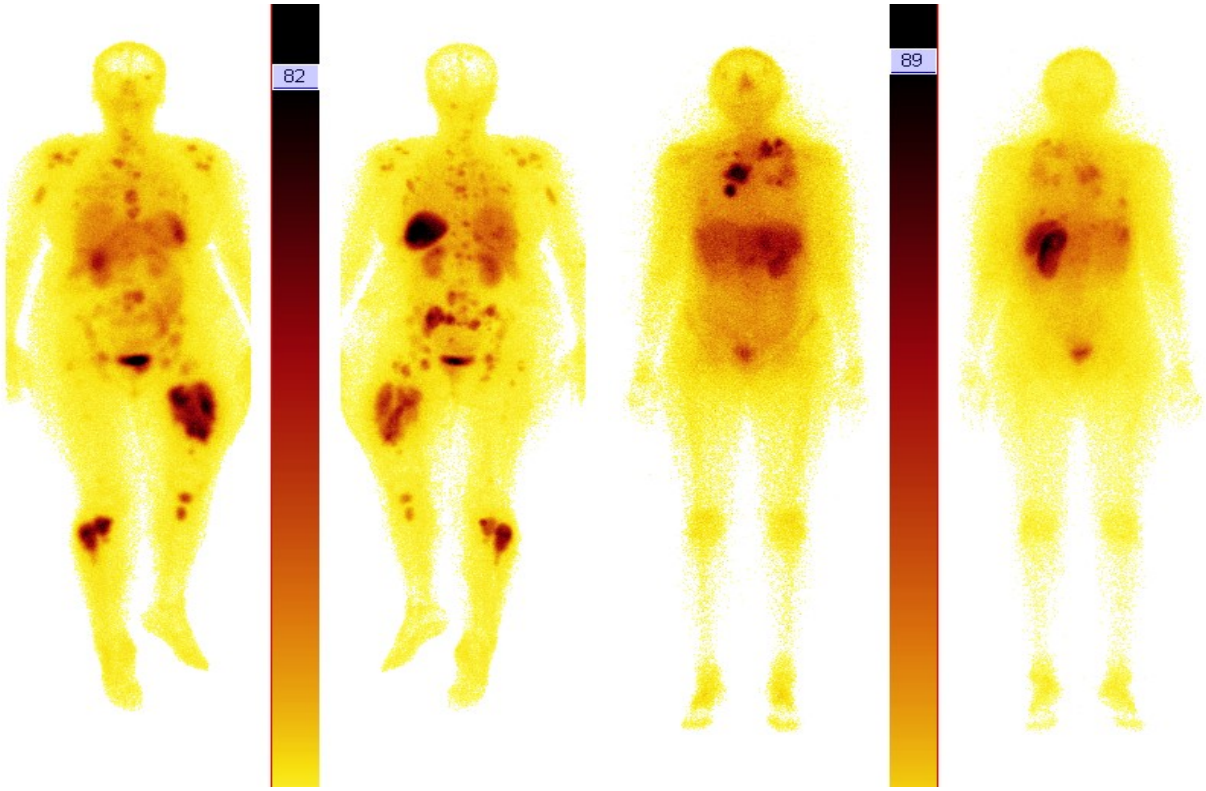


Paraganglioma Vagal et Carotid Body dex.



SOMATOSTATIN RECEPTOR IMAGING

^{99m}Tc-Tectrotyd (tyr^3-octreotyd)



pheochromocytoma

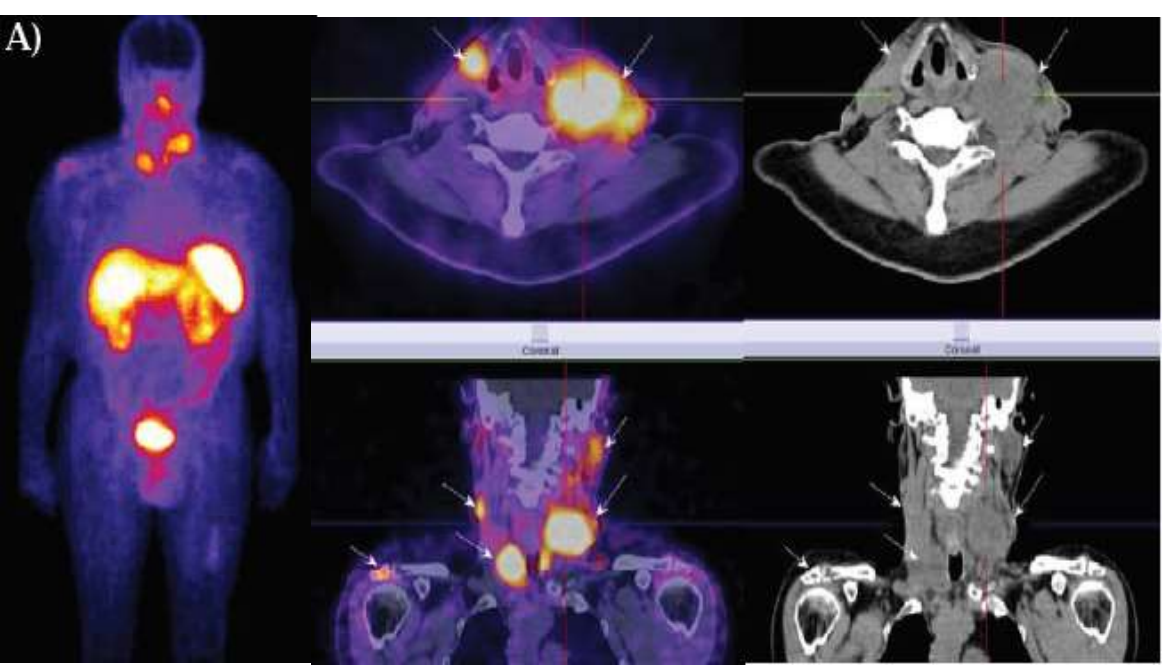
paraganglioma

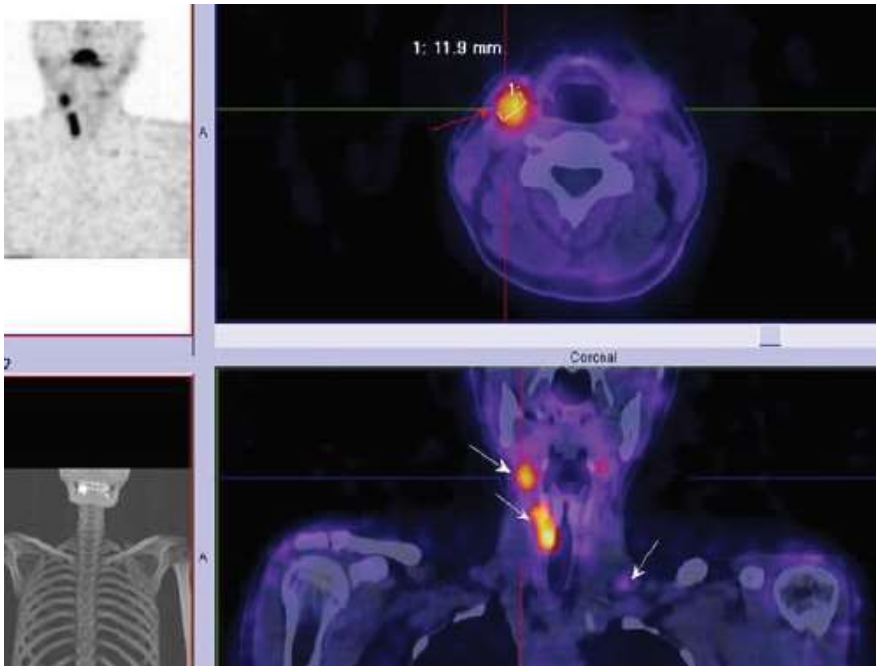
MEDULLARY THYROID CARCINOMA

- Arise from C cells $\rightarrow$ CALCITONIN, CEA, serotonin, VIP
- 80% Sporadic , or familial $\pm$ MEN Syndrome
- Composed of polygonal or spindle cells , usually with demonstrable AMYLOID in the stroma
- Calcitonin demonstrated in tumor cells
- Level of calcitonin in serum may be useful for follow up
- Family members may show C cell hyperplasia ,$\uparrow$ Calcitonin, & RET mutation
- Metastases by blood stream
- Prognosis intermediate , worse in MEN. 2B

SOMATOSTATIN RECEPTOR IMAGING

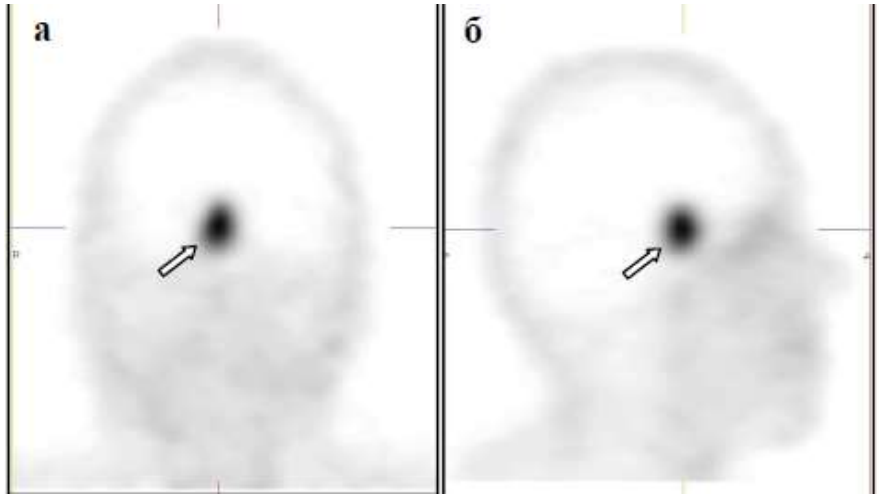
MTC (Medullary thyroid carcinoma)



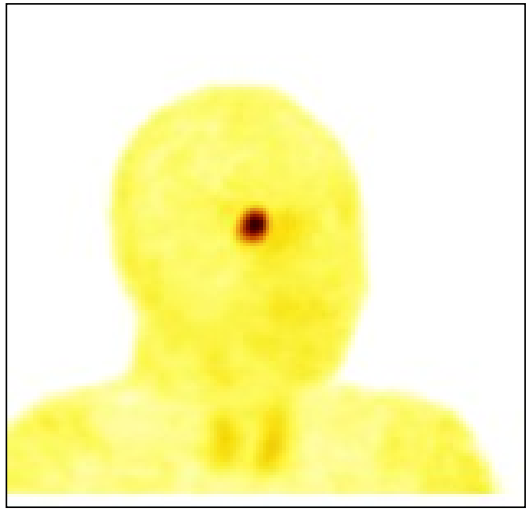


PITUITARY SCAN

^{99m}Tc-Tectrotyd (tyr^3-octreotyd)



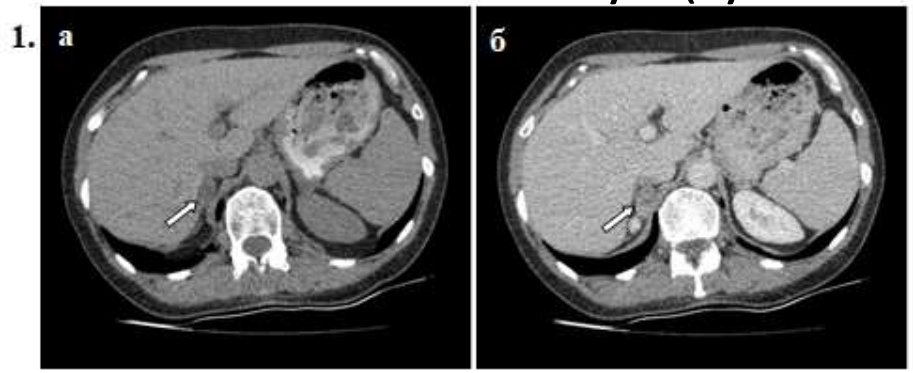
коронални пресек сагитални пресек

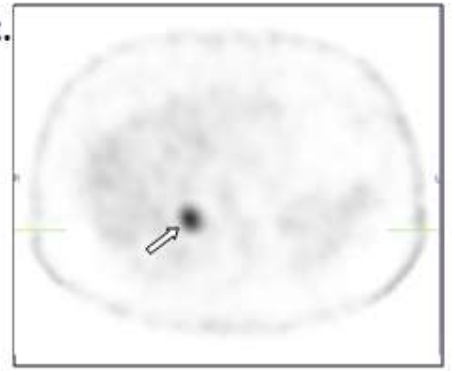


томографска реконструкција

SOMATOSTATIN RECEPTOR IMAGING

^{99m}Tc-Tectrotyd (tyr^3-octreotyd)

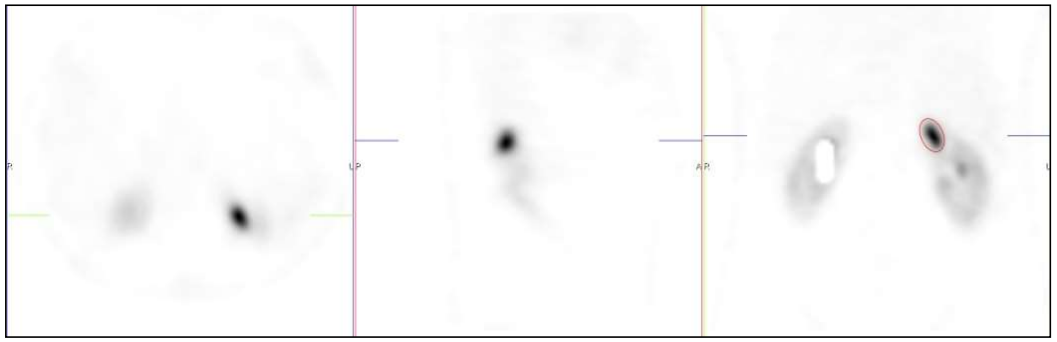


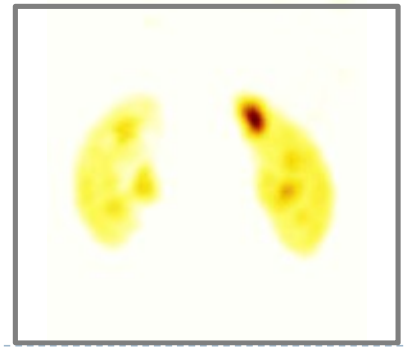


- 1) MDCT
- 2) ^{99m}Tc-Tektrotyd

^{99m}Tc-MIBI

Adenoma gl. suprarenalis dex. (Cushing Sy)





Trans. Sagital Coronal