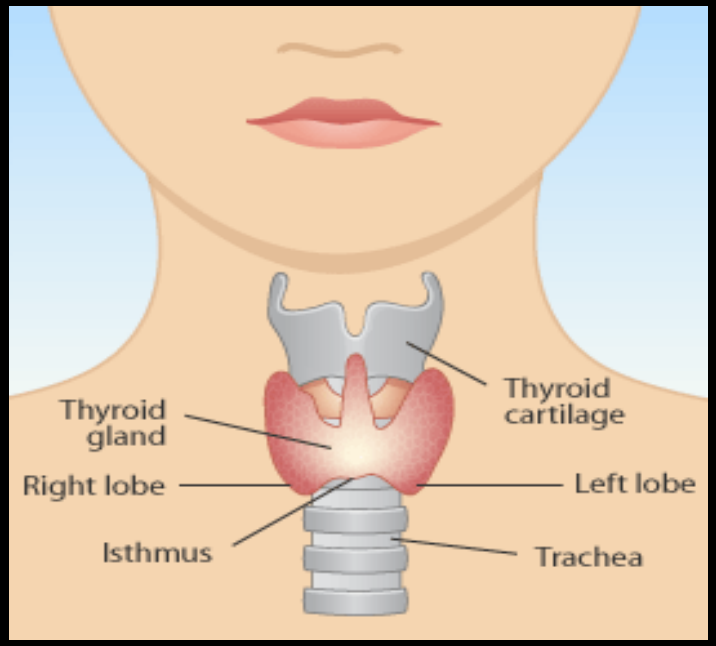
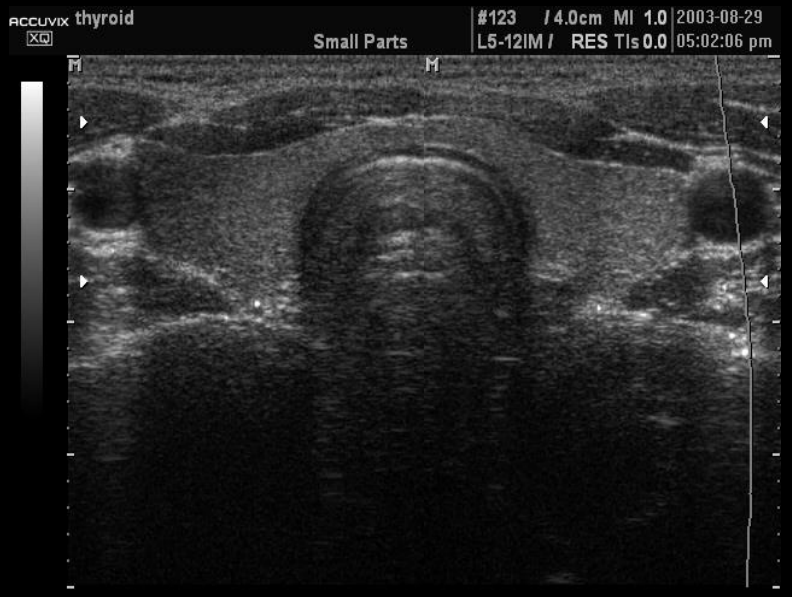
Pathology of the thyroid, and parathyroid gland(s)


Thyroid gland
Thyroid cartilage
Right lobe
Left lobe
Isthmus
Trachea

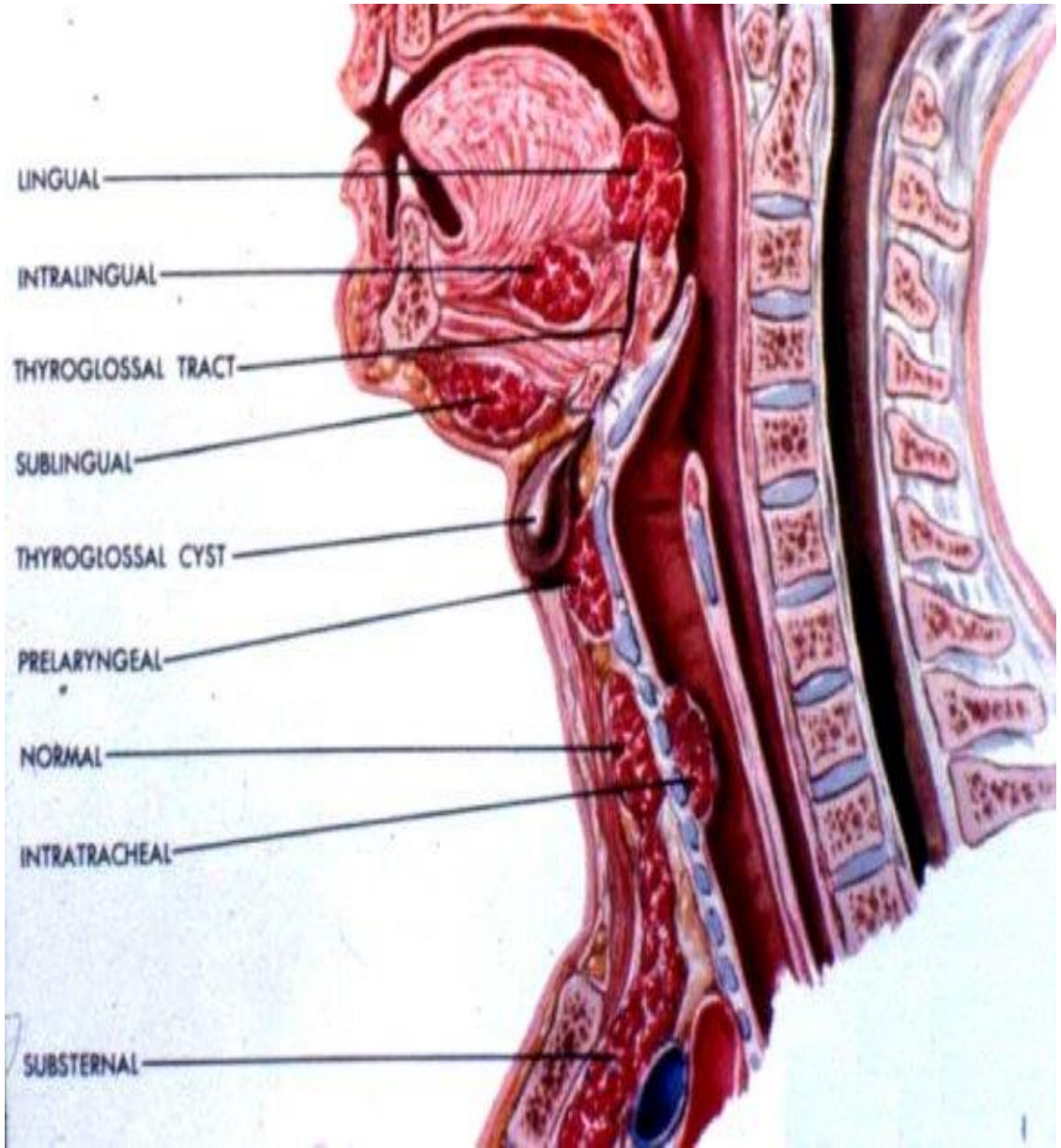

ACCUVIX thyroid
XQ
Small Parts
#123 / 4.0cm MI 1.0
L5-12IM / RES TIs 0.0
2003-08-29
05:02:06 pm

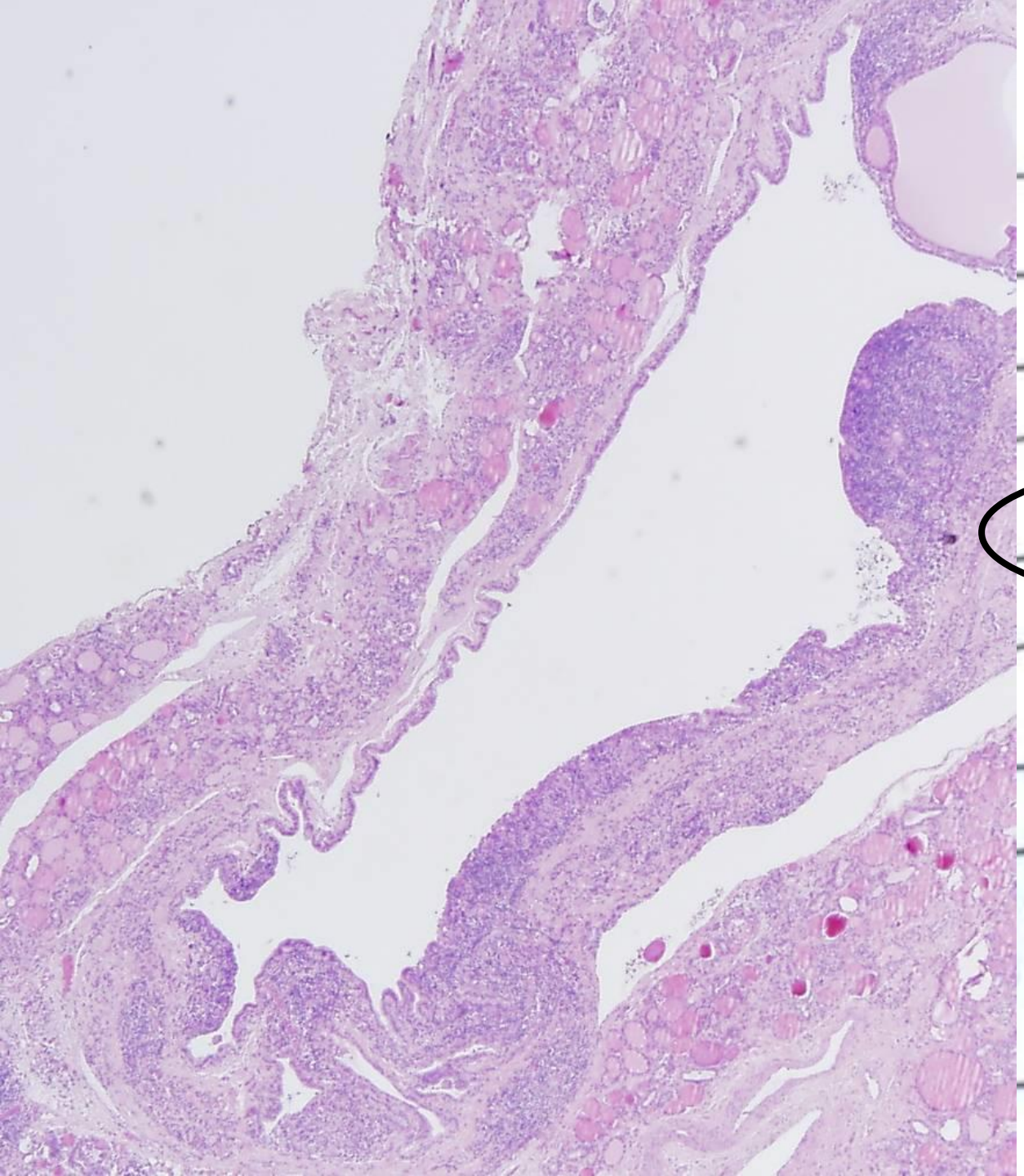
Development

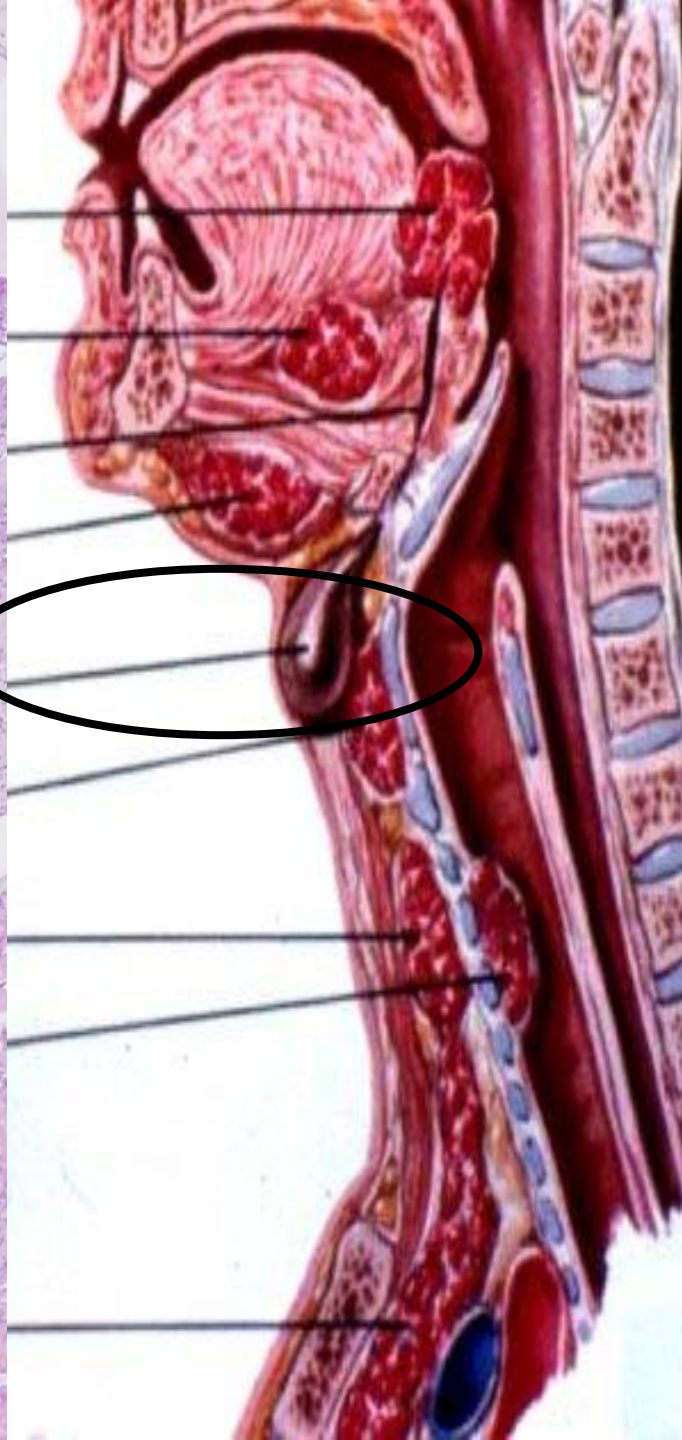
Pharyngeal epithelial pouch
(basis of the tongue)
(foramen cecum)
(Struma lingualis)

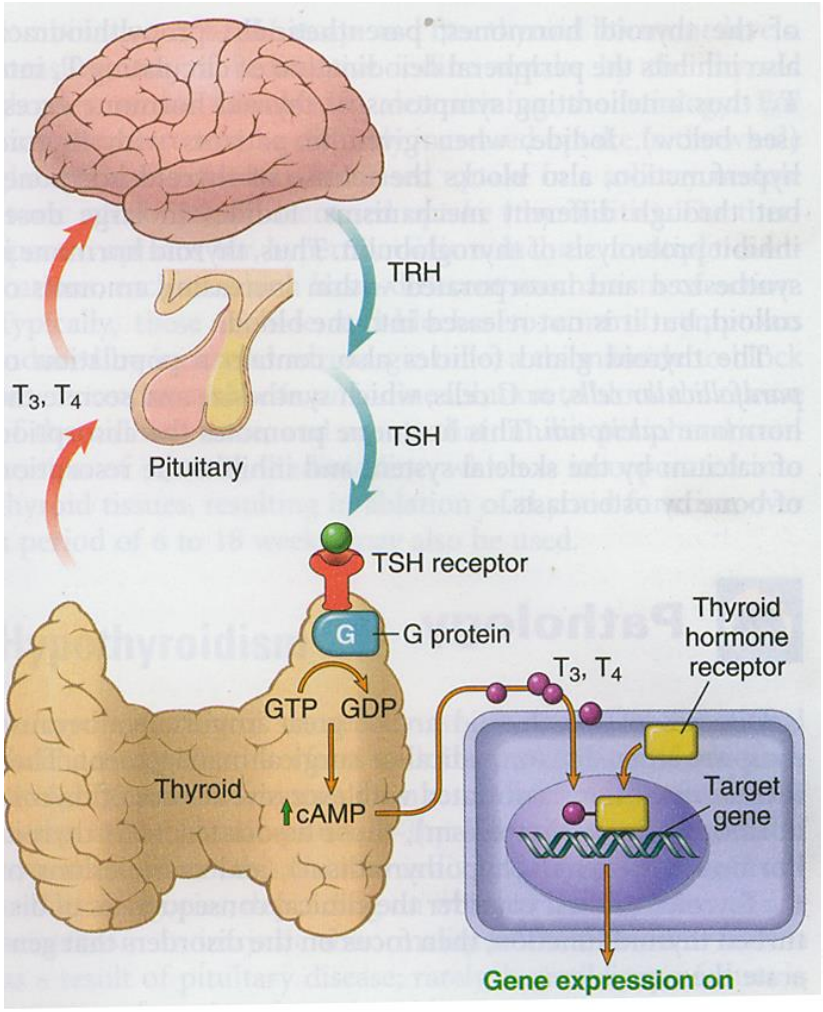
Ductus thyroglossus

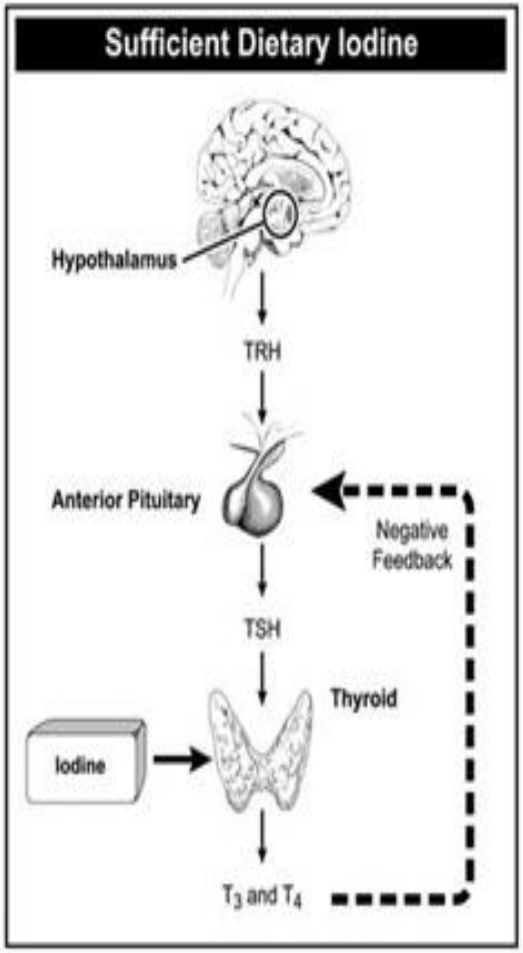
Substernal
Thyroid tissue

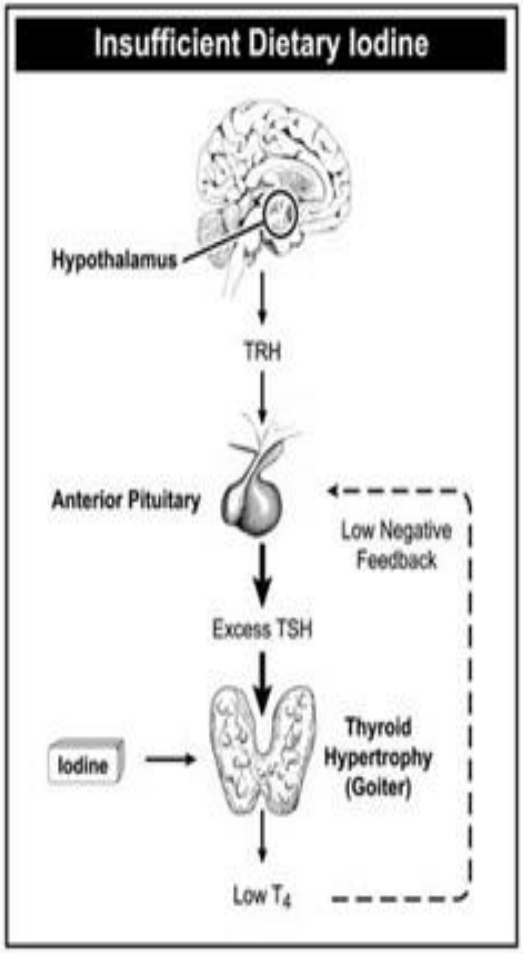


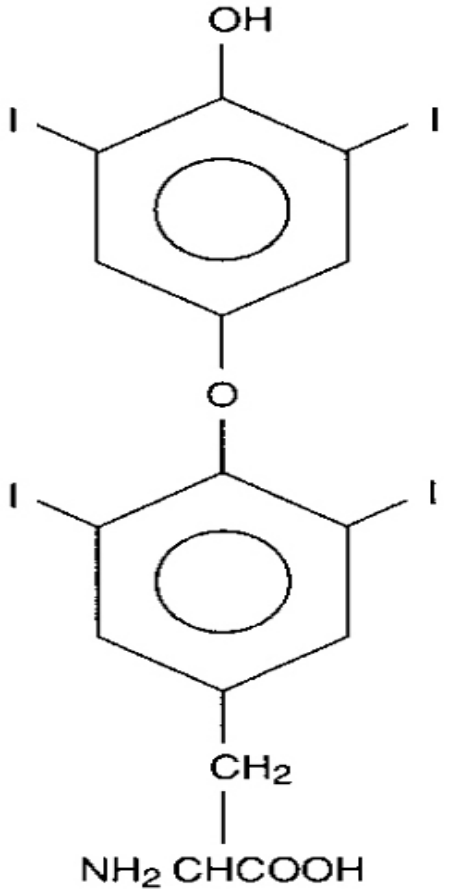




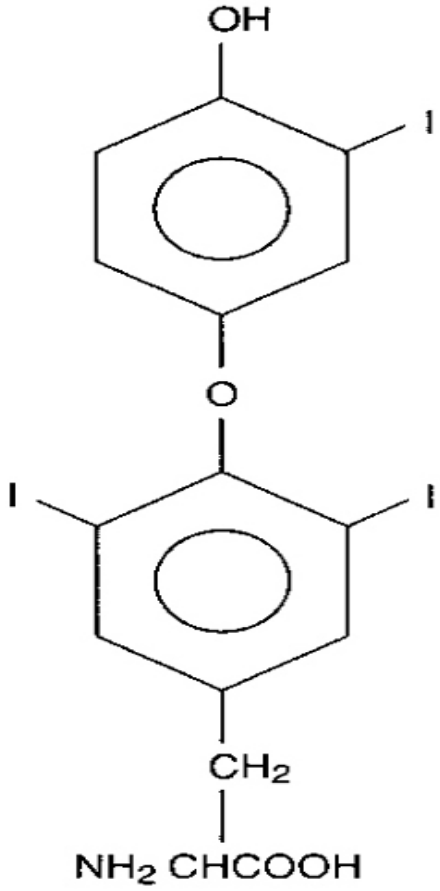

TRH
TSH
T_3, T_4
Pituitary
TSH receptor
G
G protein
GTP
GDP
Thyroid
cAMP
T_3, T_4
Thyroid hormone receptor
Target gene
Gene expression on


Sufficient Dietary Iodine
Hypothalamus
TRH
Anterior Pituitary
Negative Feedback
TSH
Thyroid
Iodine
T_3 and T_4


Insufficient Dietary Iodine
Hypothalamus
TRH
Anterior Pituitary
Low Negative Feedback
Excess TSH
Iodine
Thyroid Hypertrophy (Goiter)
Low T_4



Thyroxine



Triiodothyronine

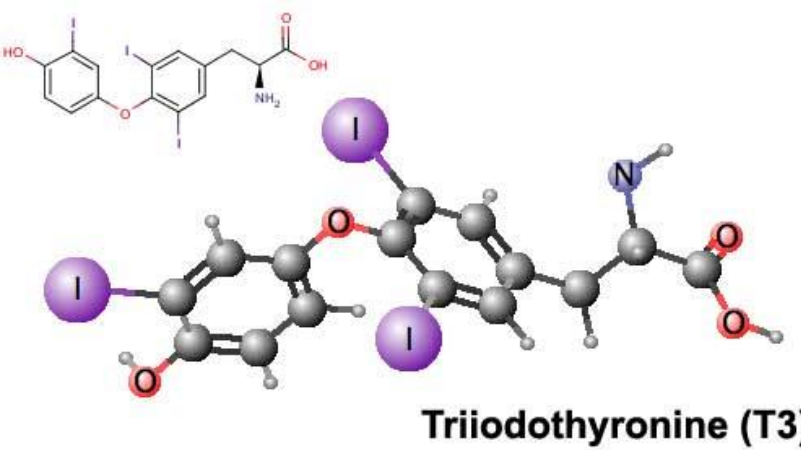
Carbohydrate catabolism,

Lipid catabolism,

Protein synthesis

} Metabolic activity ^

Brain development…



Triiodothyronine (T3)

T3, T4 in the blood:

thyroxine-binding globulin (TBG),	70%
transthyretin or "thyroxine-binding prealbumin" (TTR or TBPA)	10-15%
Albumin	15-20%
free T4 (fT4)	0.03%
free T3 (fT3)	0.3%

Nomenclature

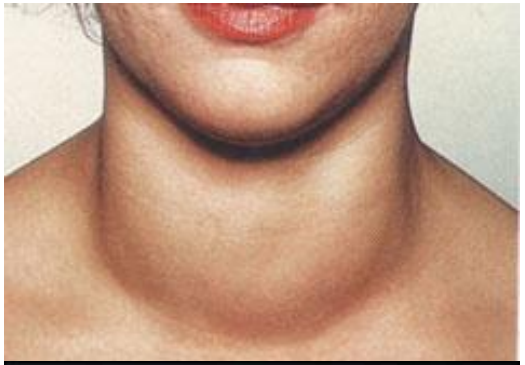
Struma diffusa
nodosa

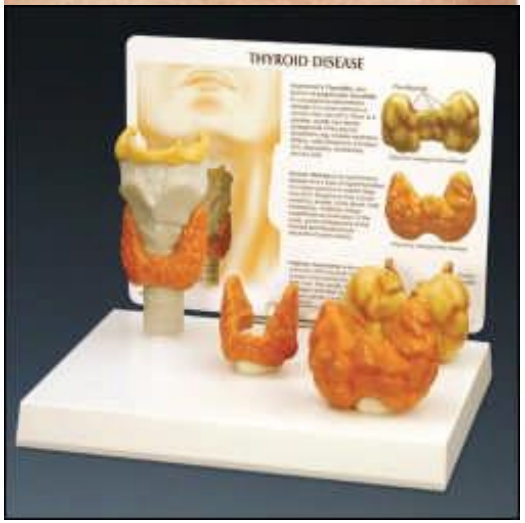
Function (?!)

Normofunction

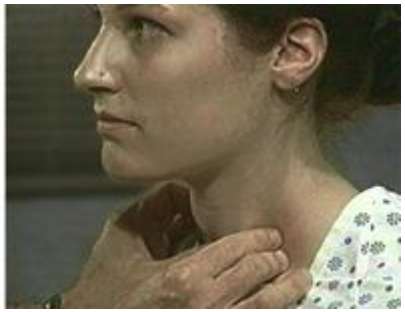
Hyperfunction

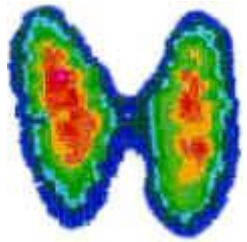
Hypofunction

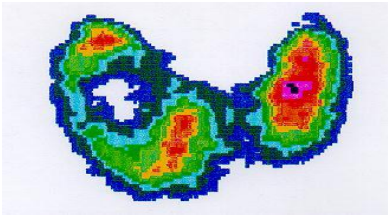


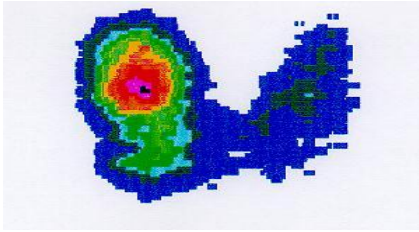


Examination of the thyroid









Physical

Laboratory TSH 0.3-3.6 mU /l

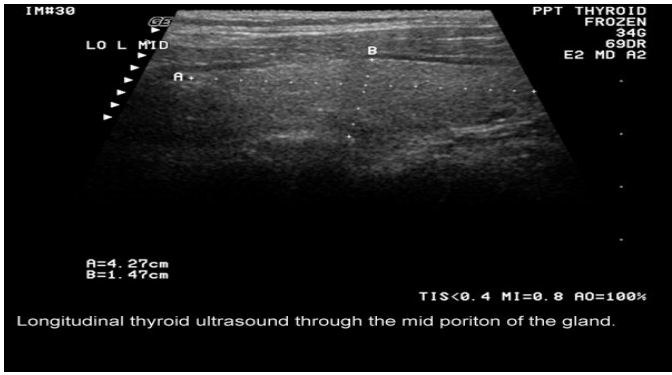
T4 9-19 pmol/l

T3 2.6-5.7 pmol/l

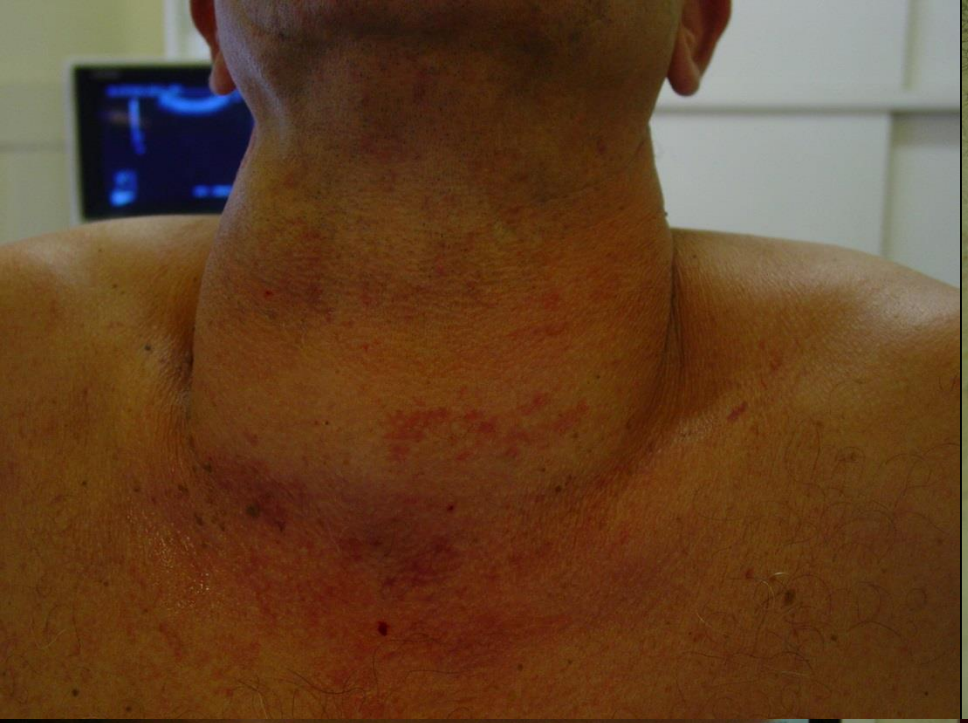
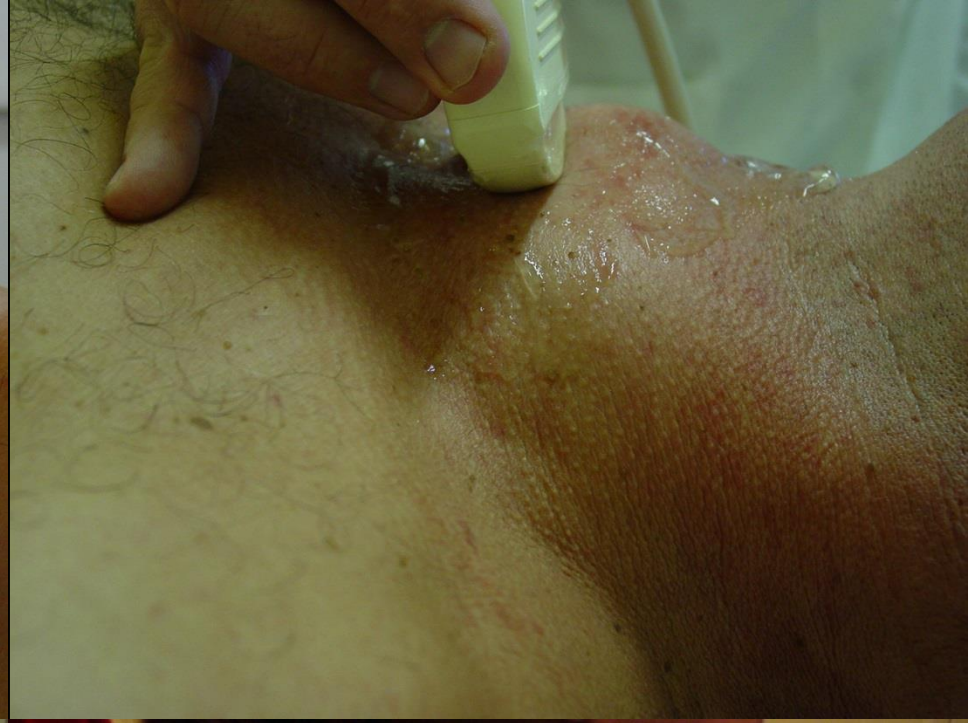
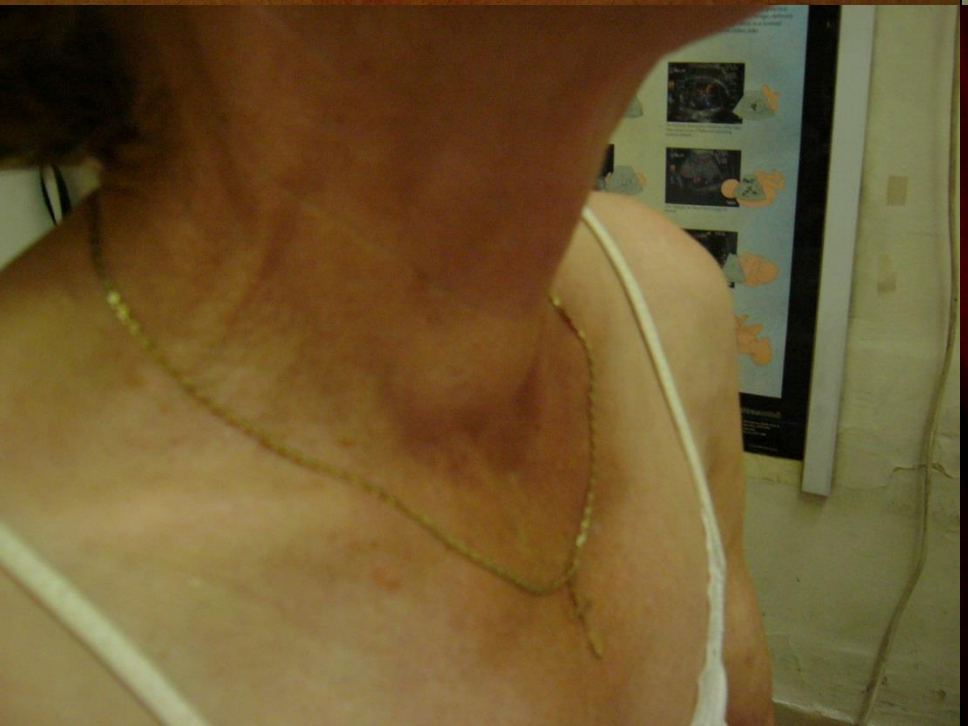
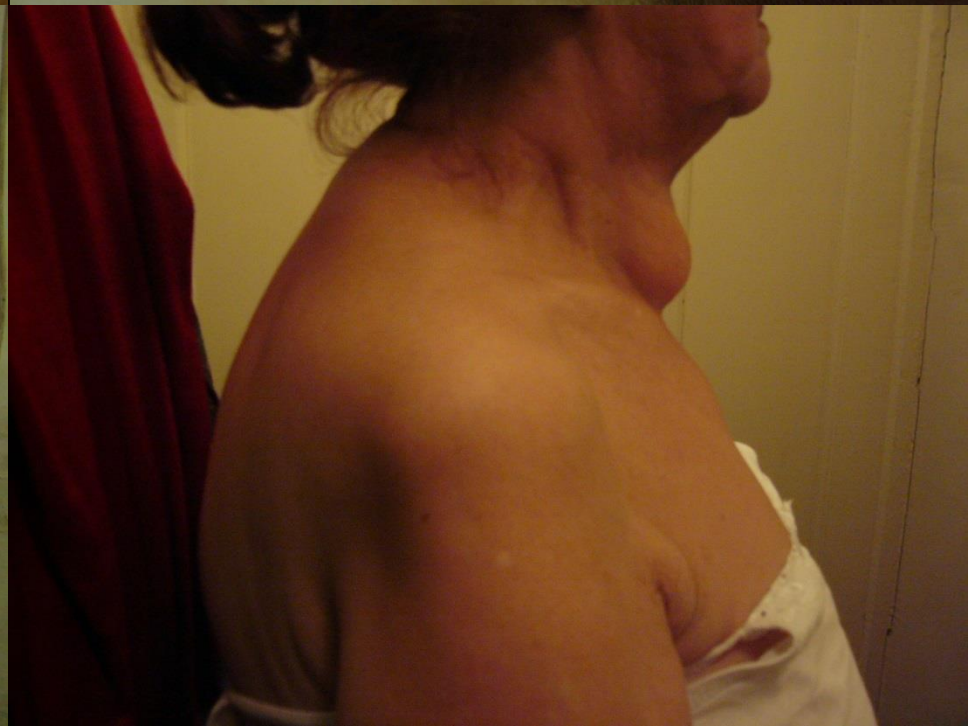
Scintigraphy

US

FNAB



Longitudinal thyroid ultrasound through the mid poriton of the gland.

TI-RADS	Interpretation	Ultrasonographicfindings
1	Normal thyroid findings	Normal thyroid tissue without any nodular aspect
2	Constantly benign aspect	• simple cyst, spongiform nodules • "white knight" • isolated macrocalficication, nodular hyperplasia
3	Very probably benign	Nosignsof high suspicion, isoechoic or hyperechoic, partial incapsulated
4A	Undetermined	Nosignsof high suspicion, mildlyhypoechoic, encapsulatednodule
4B	Suspicious	• irregularshape • tallerthanwide, irregularborders, microcalcifications, markedlyhypoechoic, high stiffnesswith elastography 1 or 2 signsandnolymphnodemetastasis
5	Highly suspicious	• irregularshape/ tallerthanwide, irregularborders • microcalcifications • markedlyhypoechoic • high stiffness with elastography: strain ratio > 4 3 to 5 signsand/orlymphnodemetastasis
Abbreviations: TI-RADS: Thyroid Imaging Reporting Data System		

2017 Bethesda System for Reporting Thyroid Cytopathology

Diagnostic Category	ROM if NIFTP not cancer	ROM if NIFTP is cancer	Management
Nondiagnostic/unsatisfactory Cyst fluid only Acellular specimen Other: Obscuring factors	5–10%	5–10%	Repeat fine needle aspiration under ultrasound guidance
Benign Benign follicular nodule Chronic lymphocytic (Hashimoto) thyroiditis, in proper clinical setting Granulomatous (subacute) thyroiditis	0–3%	0–3%	Clinical and US follow-up until two negative
Atypia of undetermined significance/ follicular lesion of undetermined significance	6–18%	10–30%	Repeat FNA, molecular testing, or lobectomy
Follicular neoplasm/ suspicious for a follicular neoplasm (Specify if Hürthle cell type)	10–40%	25–40%	Molecular testing, lobectomy
Suspicious for malignancy	45–60%	50–75%	Lobectomy or near-total thyroidectomy
Malignant Papillary thyroid carcinoma Medullary thyroid carcinoma Poorly differentiated carcinoma Undifferentiated (anaplastic) carcinoma Squamous cell carcinoma Carcinoma with mixed features Metastatic malignancy Non-Hodgkin lymphoma Other	94–96%	97–99%	Lobectomy or near-total thyroidectomy

Hyperthyreosis - effects

Sympathetic tone (ß-adrenerg tone) - basal metabolic activity- ^^

Skin: warm, wet, heat intolerance
Loss of weight, diomyopatia

Heart: tachycardy, cardiomegaly, arrhytmia (atrial fibrillation), CHF- TDC
(congestive heart failure, thyreotoxic dilatative cardiomyopaty)

Neuromuscular system:
tremor, hyperactivity, insomny, emotional lability, anxiety, proximal muscle weakness, loss of muscle

Ocular changes:
„eyes shut wide" - levator palpebrae sympathetic overdose
real exophtalm only in Graves disease

GI: hypermotility, malbsorption, diarrhea

Bones: osteoporosis due to enhanced resorption, brittleness ^ ^

Hyperthyreosis – laboratory tests

TSH (low, even in subclinical stages!)

T4 level

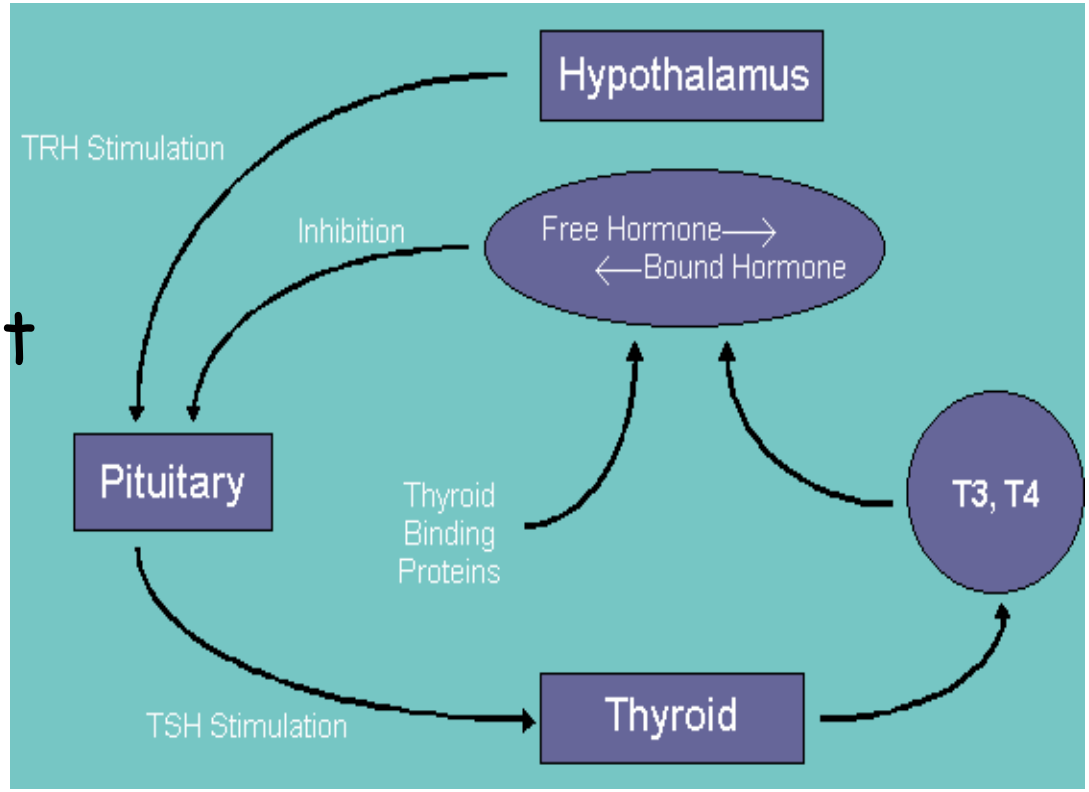
T3 level

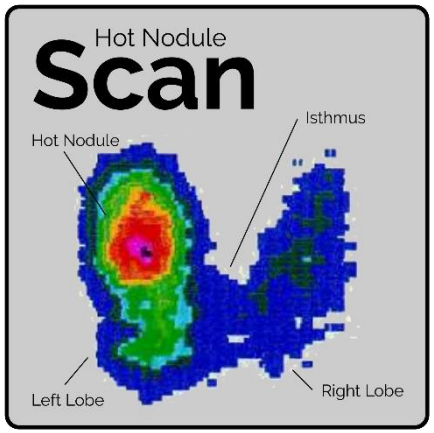
Free

T4, T3 measurement

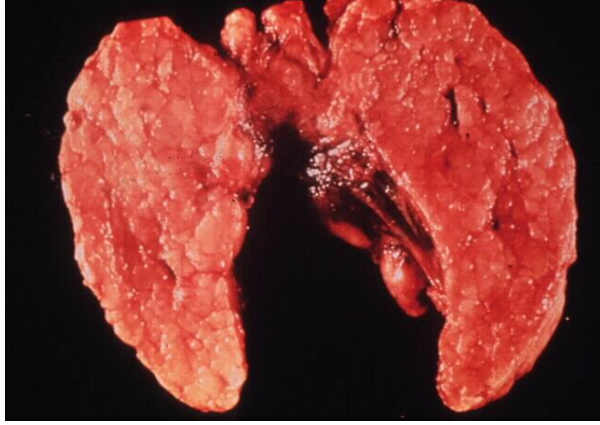
TRH test

Scintigraphy





Thyreotoxicosis - causes



Hyperthyreosis

Primary

- Diffuse toxic hyperplasia (Graves- Basedow)
- Toxic multinodular goiter
- Toxic adenoma
- Thyroid carcinoma
- Neonatal hyperfunction (maternal Graves)

Secondary

- Hypophysis adenoma

Thyreotoxicosis – causes
Non-Hyperthyreotic states

De Quervain thyreoiditis (Subacute granulomatous thyreoiditis)

Subacute lymphocytic thyreoiditis

Struma ovarii

Exogenous hormone overdose

Hyperthyreosis - Therapy

Lowering of ß-adrenerg tone (ß blockers)

Propylthiouracil (hampers I oxidation, T4 synthesis, and the T4-T3 conversion in tissues)

Thiamazole (inhibits the enzyme thyroperoxidase, which normally acts in thyroid hormone synthesis by oxidizing the anion iodide (I^-) to iodine (I_2))

Jodine

hampers release of stored hormone

Radiojodine therapy

destroys thyroid tissue

Hypothyreosis - Cretenism

In case of maternal hypothyreosis in early pregnancy severe

Later – less severe

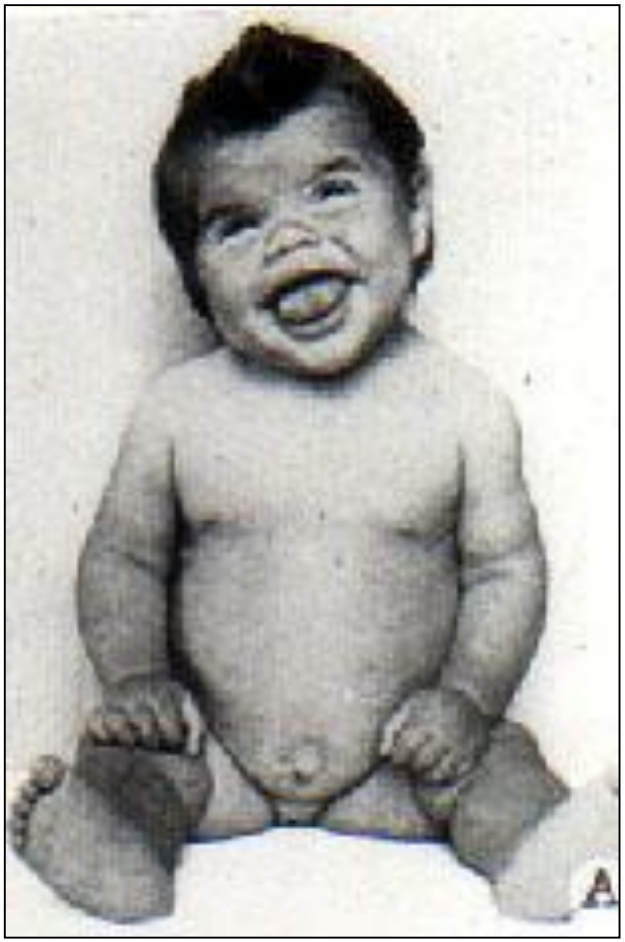
Impaired development of the skeleton, CNS

Short stature

Coarse facial features

Protruding tongue

Umbilical hernia



Hypothyreosis - causes

Primary

- Developmental anomaly (thyroid dysgenesis: PAX-8, TTF2, TSH-R mut.)
- Thyroid hormon resistance (TRß mutation)
- Congenital biosynthetic defect (dyshormonogenetic goiter)
- Postablation

  (operation, radiojodine th, irradiation)
- Autoimmune thyroiditis
- Iodine deficiency
- Drugs (lithium, PAS)

Secondary

- Pituitary failure

Tertiary

- Hypothalamic failure

Hypothyreosis - Myxoedema

Slowing of physical and mental activity -similar to depression

Cold intolerance

Gain of weight

Obstipation

Decreased sweating

Reduced cardiac output

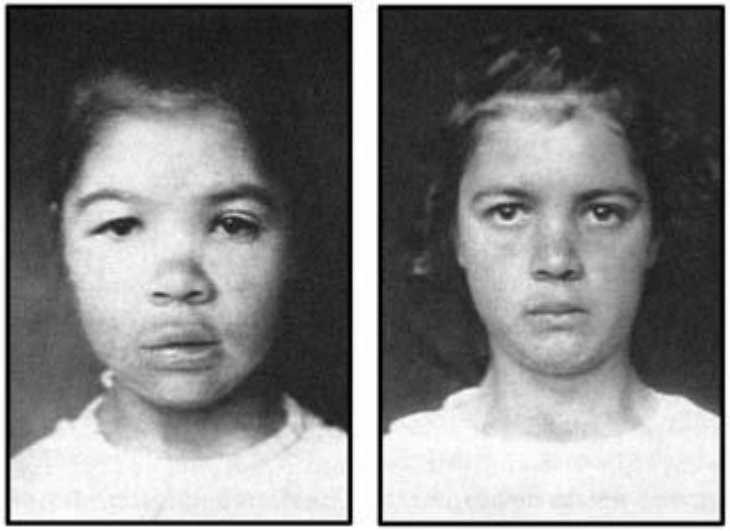
(Low output failure)

GAG, HA accumulation, oedema

Lab: TSH^^^, T3, T4ˇˇˇ,

Except for hypophysis,

hypothalamic origin



Thyreoiditis

Infectiosus

Hashimoto (chronic lymphocytic thyroiditis)

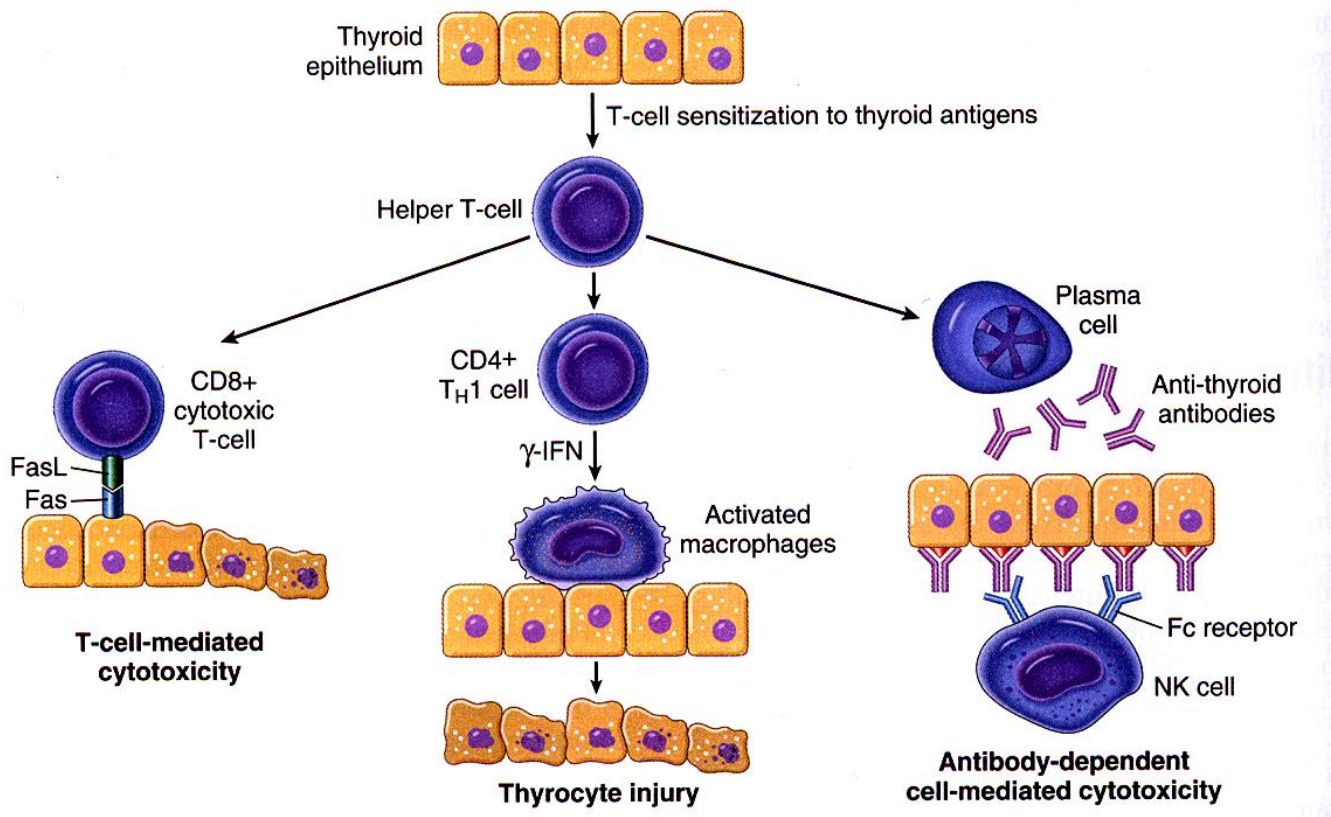
Subacute granulomatous thyroiditis – De Quervain

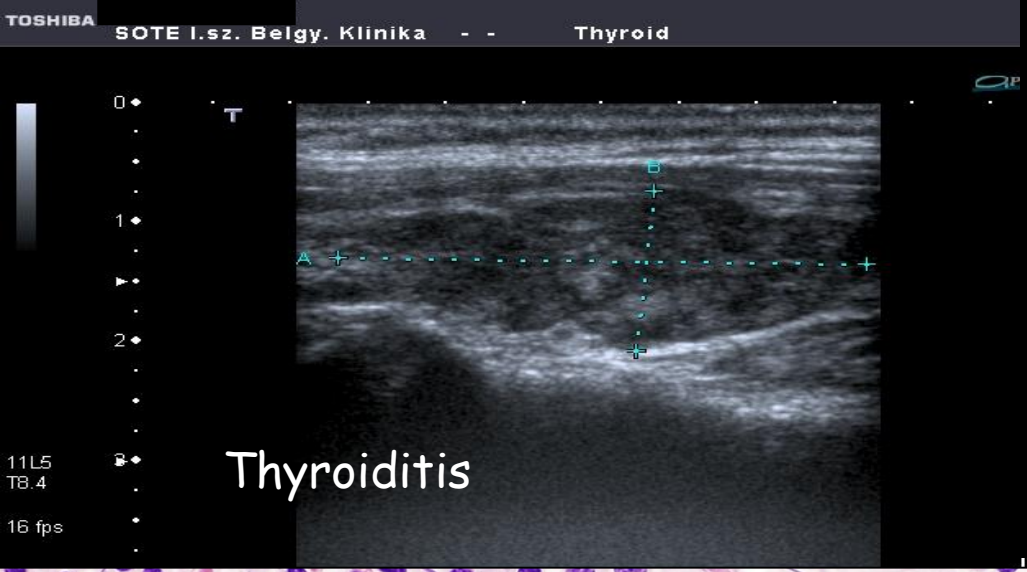
Subacute lymphocytic thyroiditis

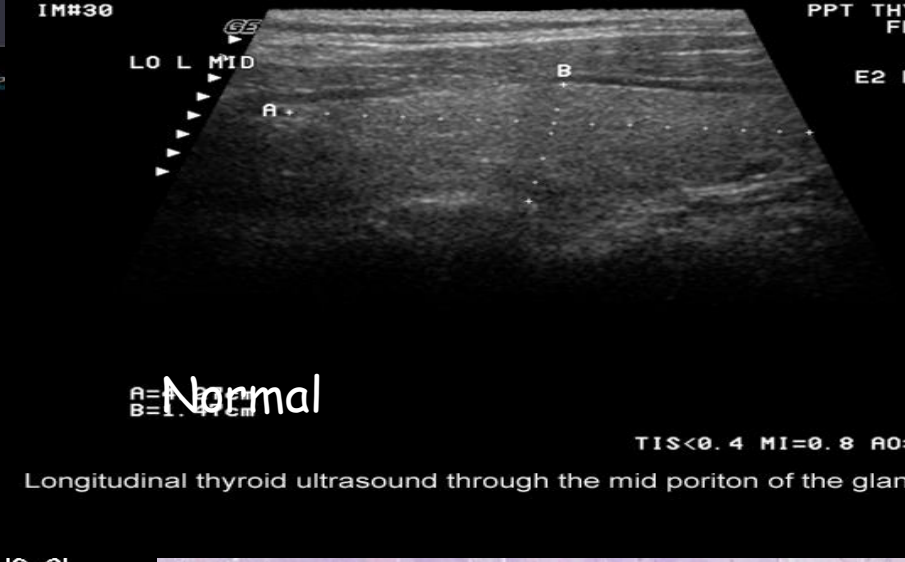
Riedel goiter

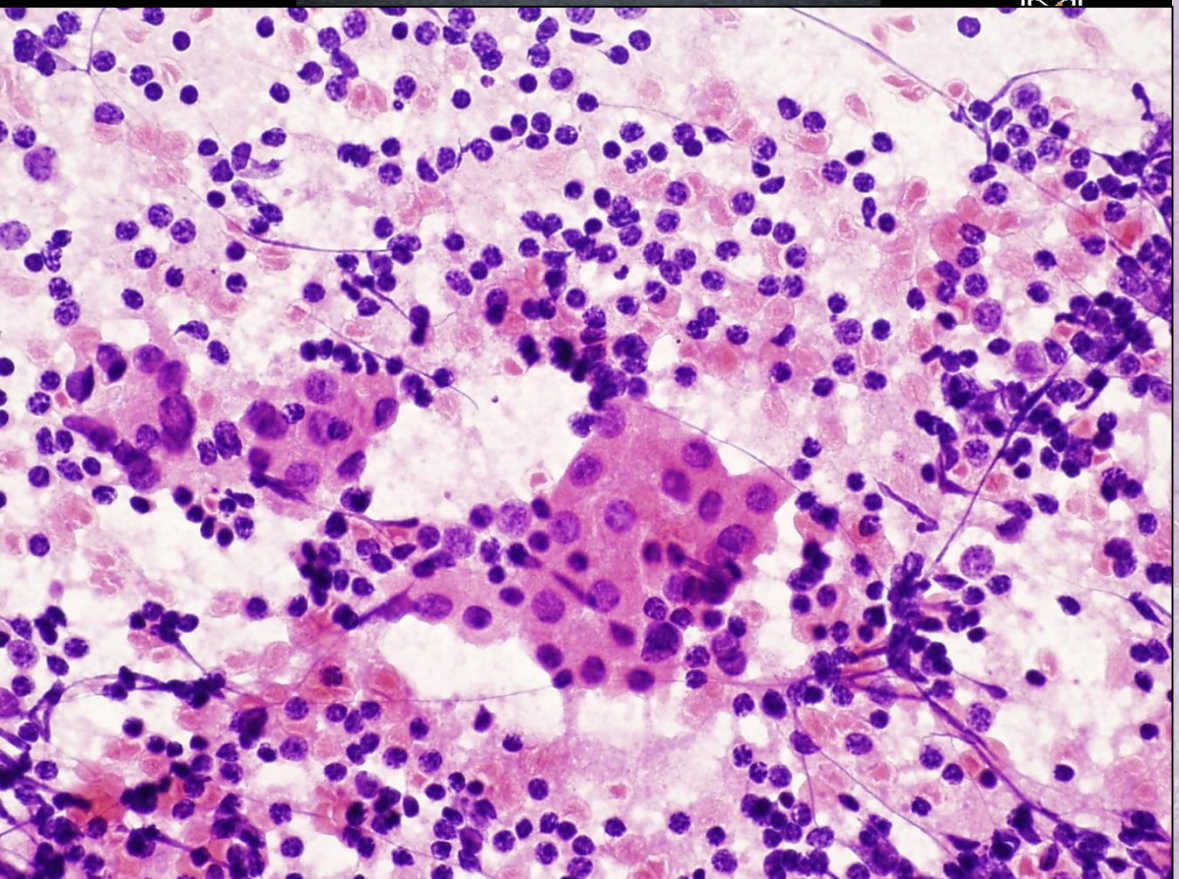
Hashimoto

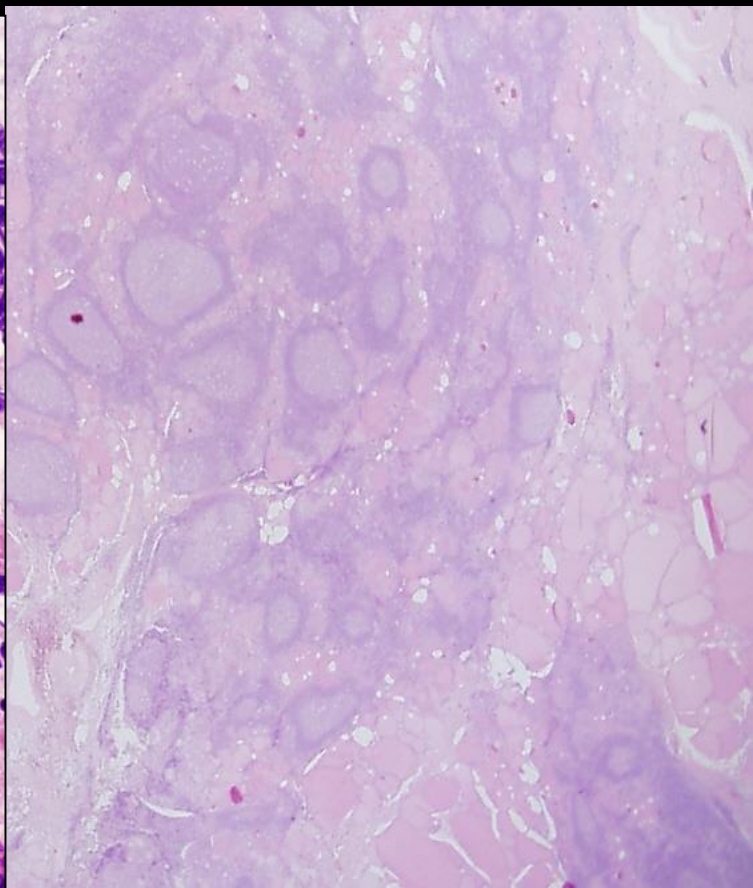
Inheritance? (Monozigotic twins 30-60 % concordance)
HLA-Dr3, HLA-DR5, polymorphism,
6p, 12q – susceptibility locus

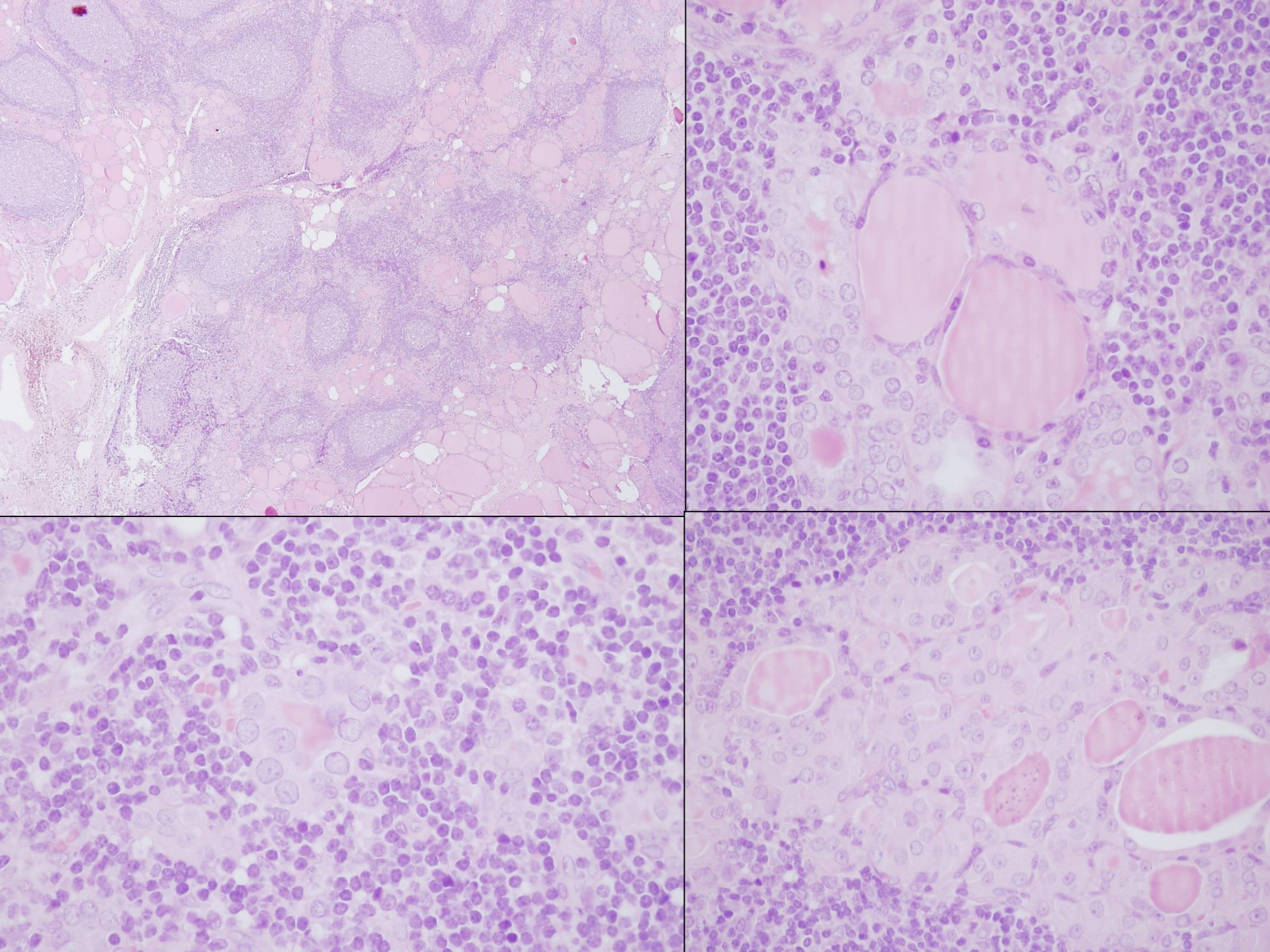



TOSHIBA
SOTE I.sz. Belgy. Klinika - - Thyroid
Thyroiditis
11L5
T8.4
16 fps


IM#30
LO L MID
Normal
TIS<0.4 MI=0.8
Longitudinal thyroid ultrasound through the mid poriton of the glan







Clinical course

Hyper (in early stage), more frequently

Hypothyreosis

Painless diffuse thyroid enlargement

(may be localised, or nodular)

T3, T4 ˇ, TSH ^ ^ ^, anti TPO

May be associated with other autoimmune diseases:

Diabetes I., Autoimmune adrenalitis, SLE, myastenia gravis, Sjögren,

Possible consequence: NHL!

(not associated with epithelial tumors …(?))

Subacute lymphocytic thyroiditis

Rare

Pathogenesis is not clear, but may have autoimmune origin (autoantibodies might occur, but not always!)

May be the precursor of Hashimoto (not obviously!)

Frequently associated with pregnancy (postpartum thyreoiditis, may recur in repeated pregnancies)

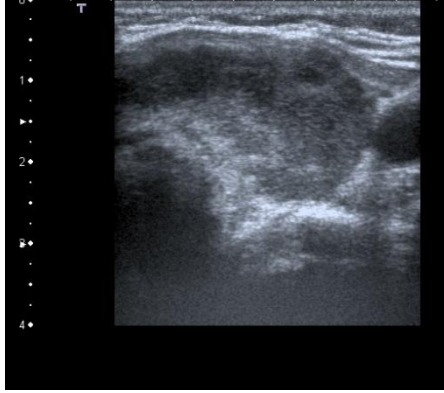
Clin.: painless thyroid enlargement, thyreotoxicosis, T3, T4 ^, TSH ˇ,

diminishing in 2-6 weeks,

After appr. 8 weeks, normal thyroid function returns

Some cases may evolve to chronic hypothyreosis

Subacute granulomatous thyroiditis – De Quervain



Postviral inflammation, -upper airway inflammation Coxsackie, mumps, measles, adenovirus

causes the release of

(viral, or thyroid originated)

AB release

Cytotoxic T cells

After the cessation of the AB release, the process is ended

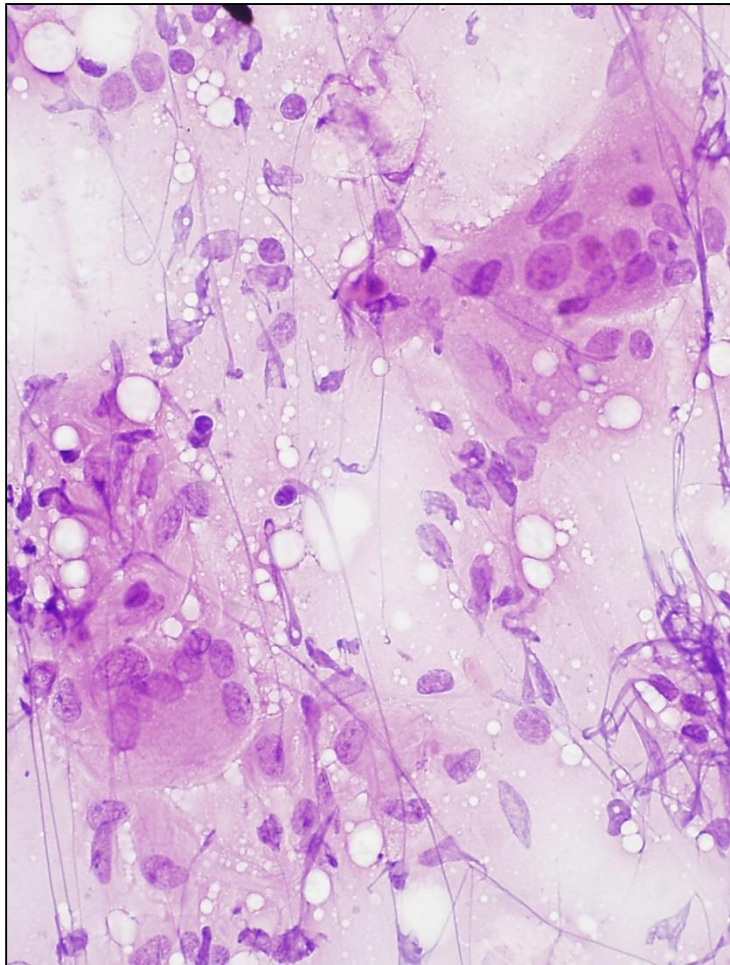
Clin.: severe cervical pain

Hyper, - than hypothyreosis,

TSH ˇ, T3, T4 ^,

scintig: low uptake

Subsides in 6-8 weeks



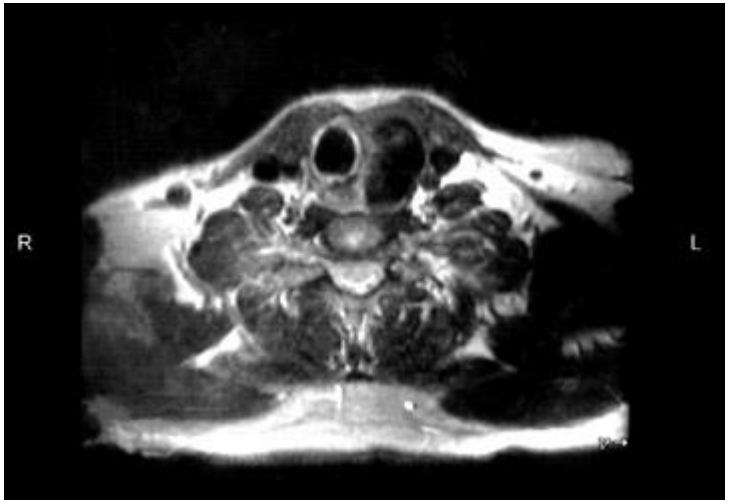
Riedel thyroiditis

Stony hard, fixed thyroid mass,

Clinically mimicking thyroid malignancy

„Burnt out", fibrotic thyroid mass

Etiology: (???), vs autoimmune



Palpation thyreoiditis

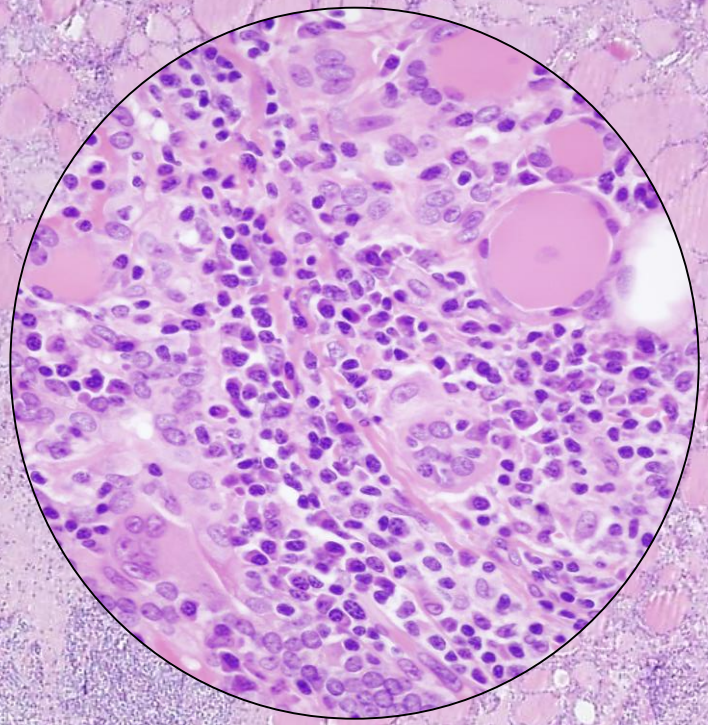
???

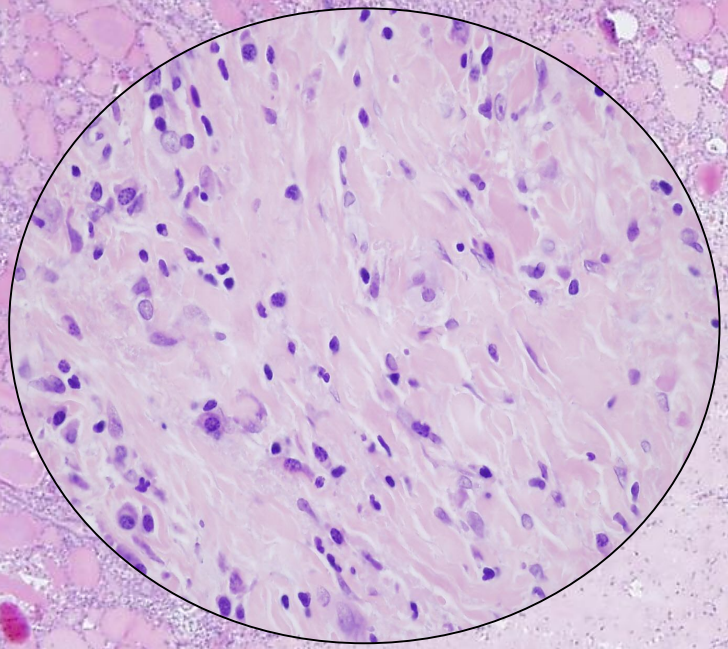
Hashimoto????

Present concept: IgG4 disease

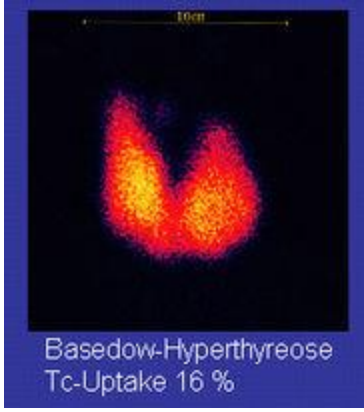
9297/07

48 y old female





Graves - Basedow



Genetics

concordance between monozygotic twins is: 60%

more frequent in certain HLA-DR 3, HLA-B8 types

CTLA-4 polymorphism (~ blocks the formation of autoantiboidies)

Autoantibodies:

Anti-TG, anti-T peroxisome, anti – TSH receptor

TSI (LATS), (this is Graves-specific)

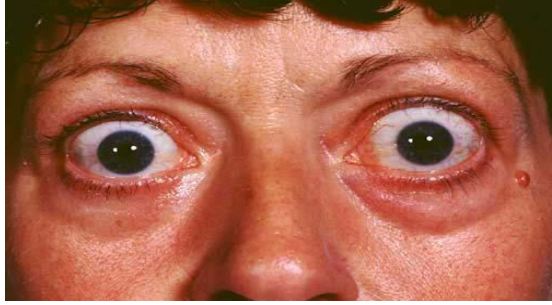
TGI

TBII (TSH-binding inhibitory immunoglobine) – this is blocking, or stimulating)

TRIGGER ?? (loss of T cell tolerance)

Anti-TG, anti-T peroxisome

Graves - Basedow



Clinical course

- Hyperthyreosis
- Exophtalmos (retroorbital ly, oedema, GAG, HA accumulation,)
- Pretibial myxoedema

Associated with other autoimmune diseases:

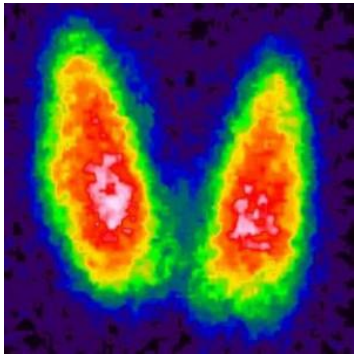
Diabetes I., Autoimmune adrenalitis, SLE, myastenia gravis, Sjögren, Anaemia perniciosa, + Hashimoto !!!!!

Lab:

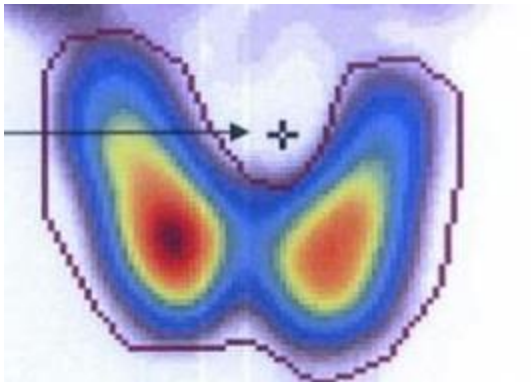
TSHˇˇˇ

T3, T4 ^^^

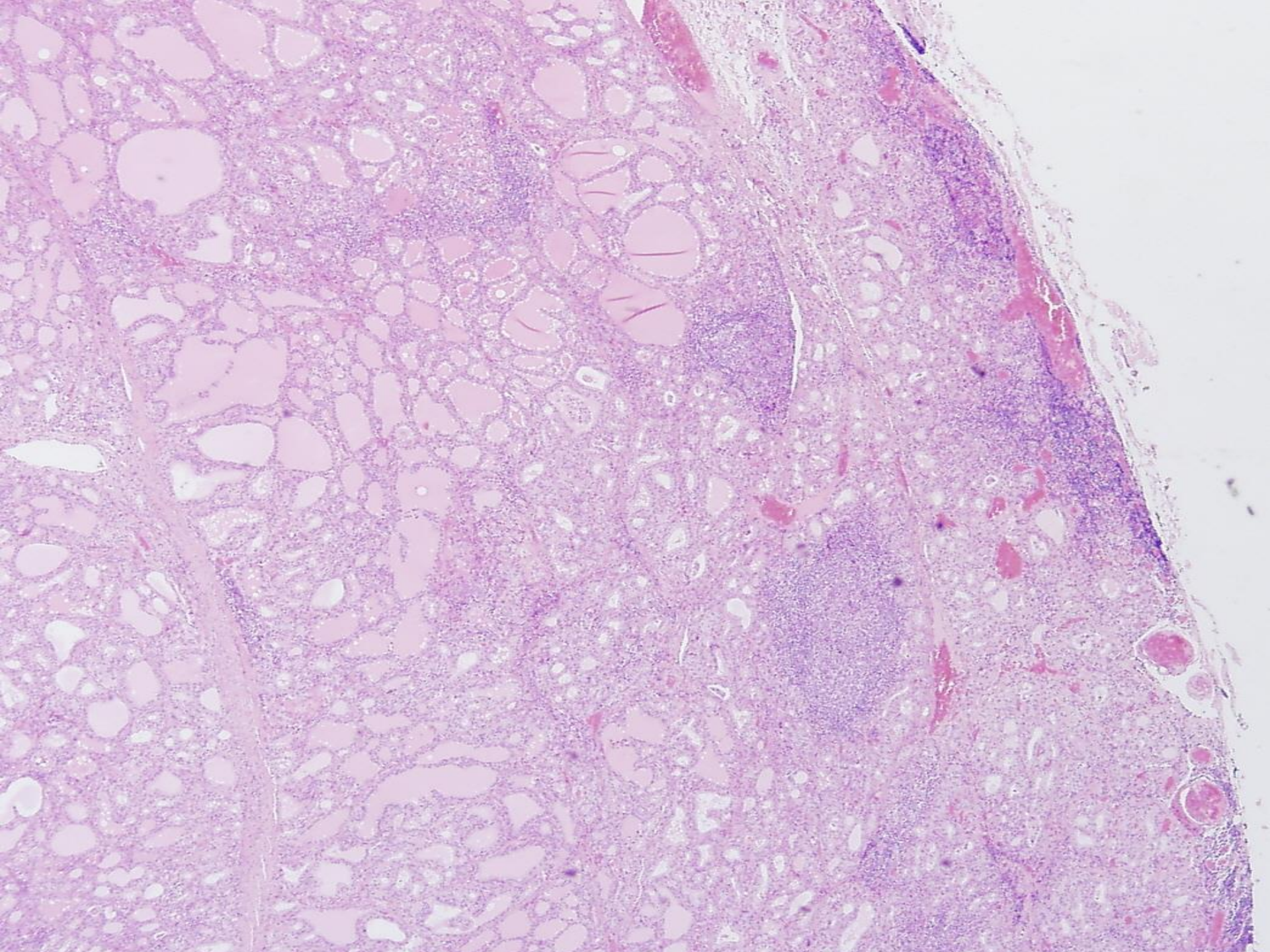
Scinti: ^^^

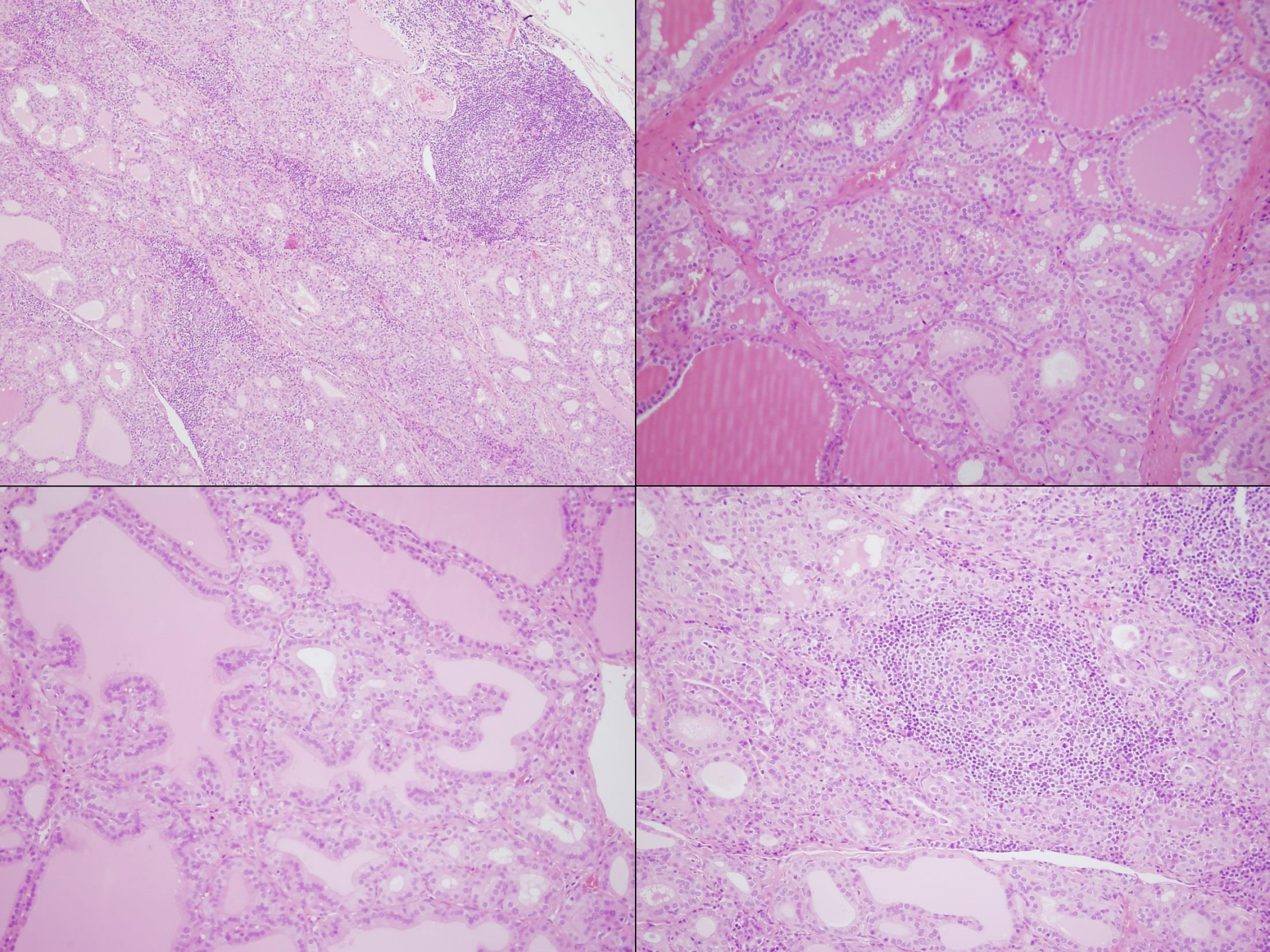


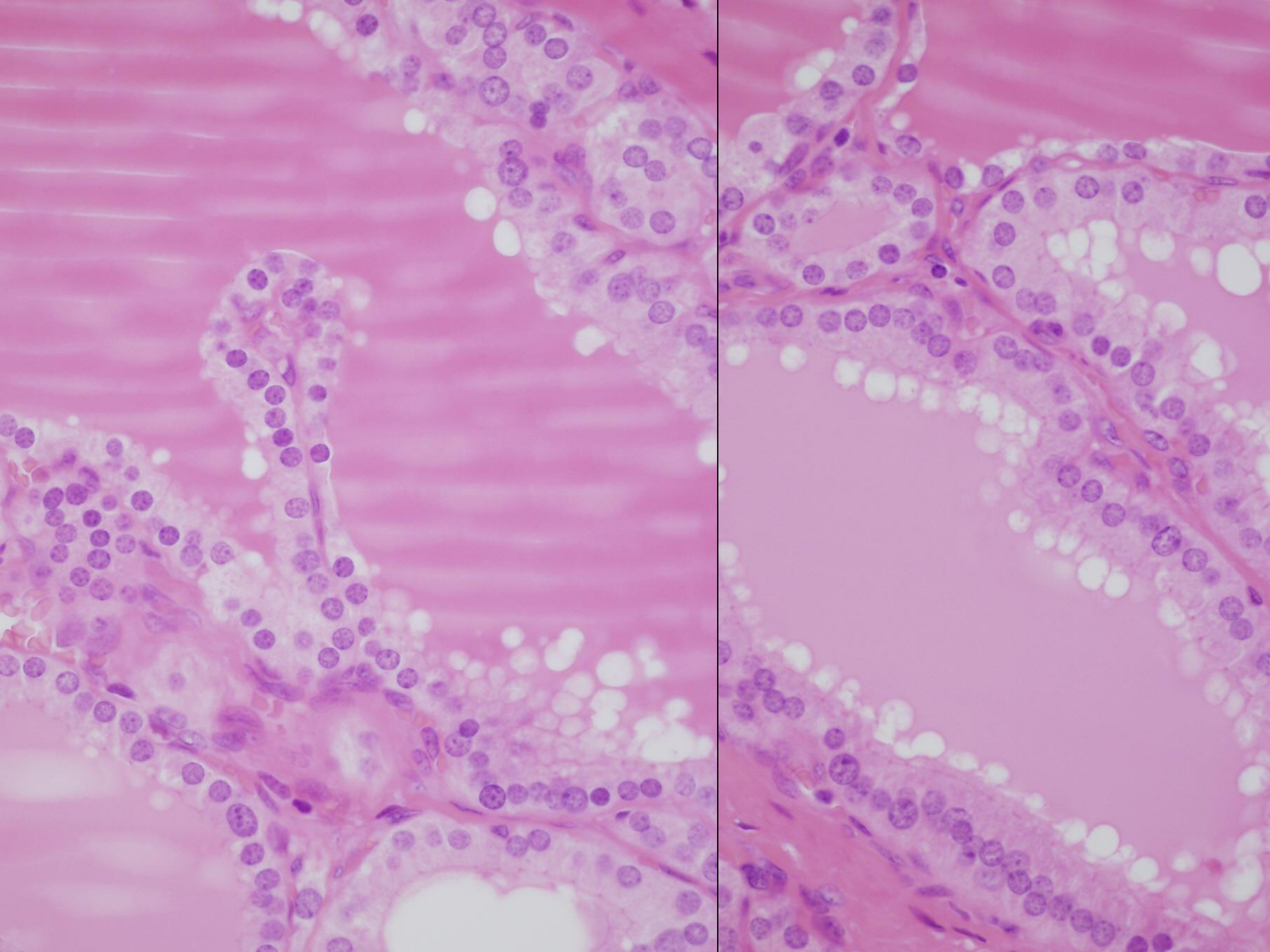
Compared to

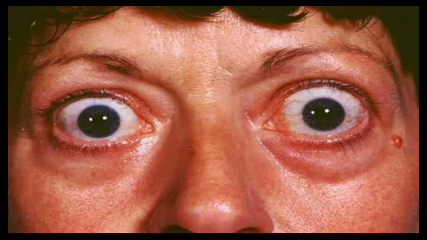


Th.: propylthiouracyl, radioiodine ablation, surgical



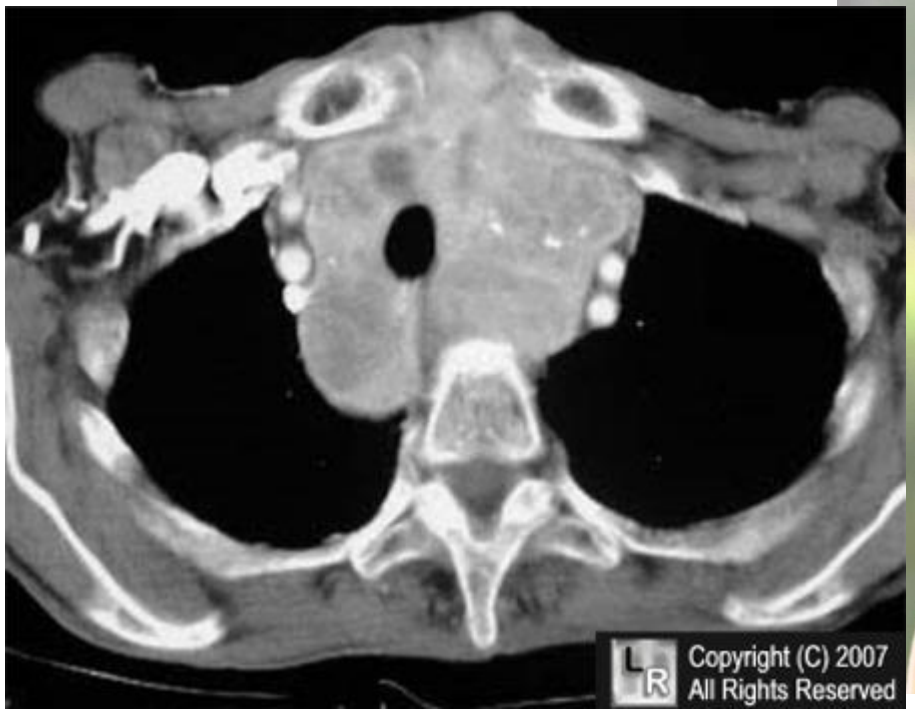






Graves ——————————— Hashimoto

Diffuse/ nodular goiter



Diffuse goiter

Endemic

(most frequent) (10 % of the population is involved)

Alps, Andes, Himalaya

Goitrogens: cabbage, cauliflower, Brussels sprouts, turnips, cassava

Iodine deficiency

Hpl, htr – euthyreoid

hypothyreoid

T3, T4 norm., TSH elevated, or upper range of normal

Sporadic

hereditary enzimatic defects

frequently unknown etiology

Nodular goiter

All longstanding simple goiters convert into ~

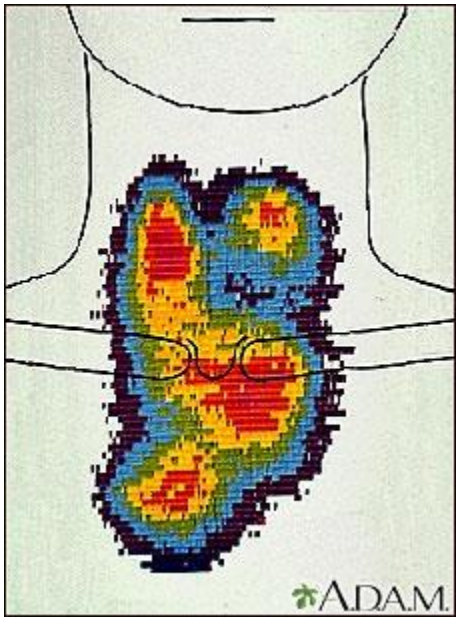
One nodule might become autonomous

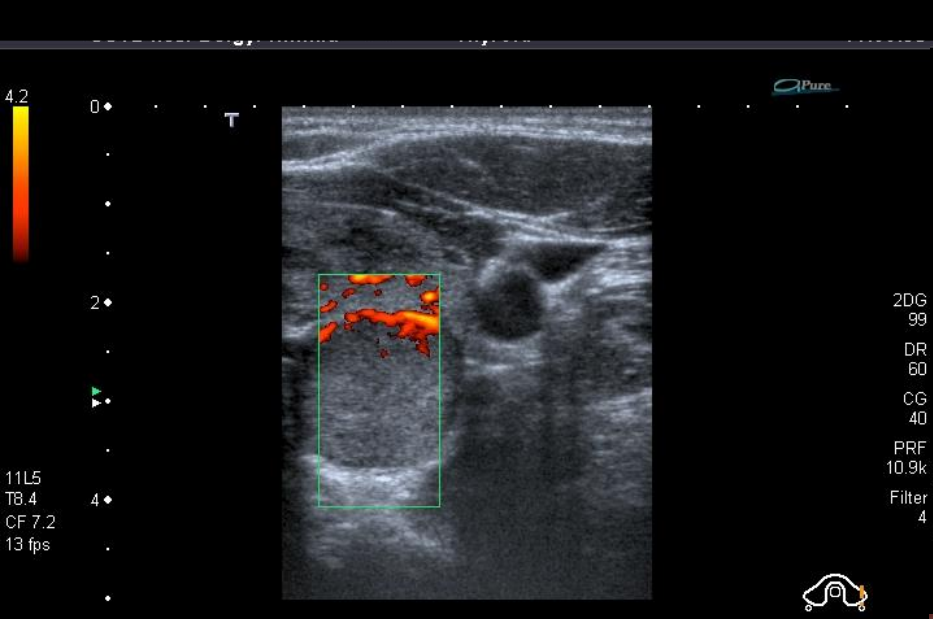
Hpl, atrophy, fibrosis, calcification, cyst formation

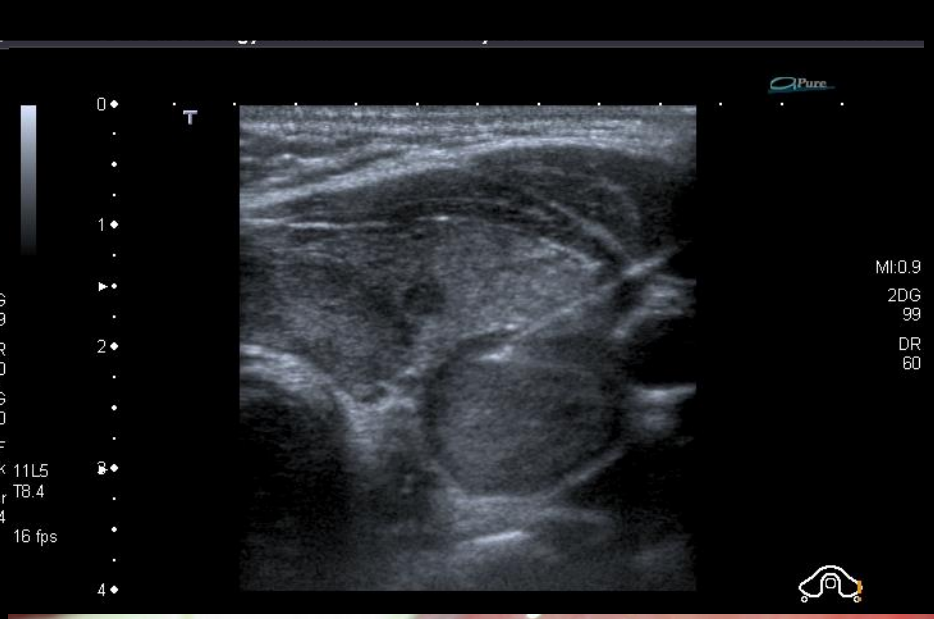
Scintigr.: uneven uptake

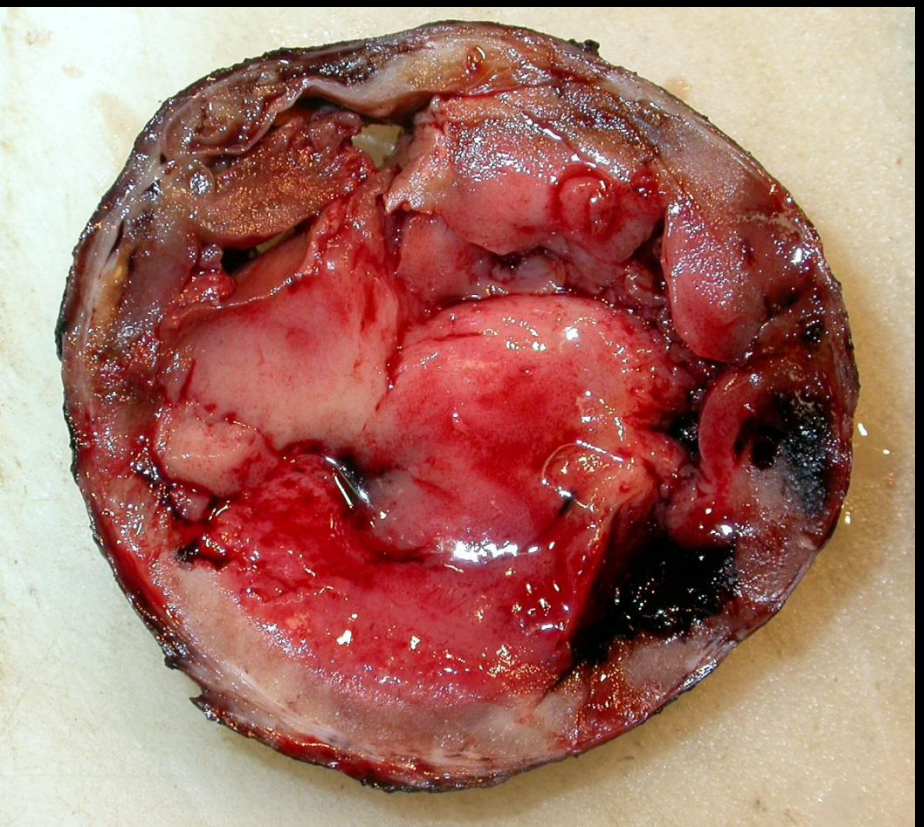
toxic nodular goiter –

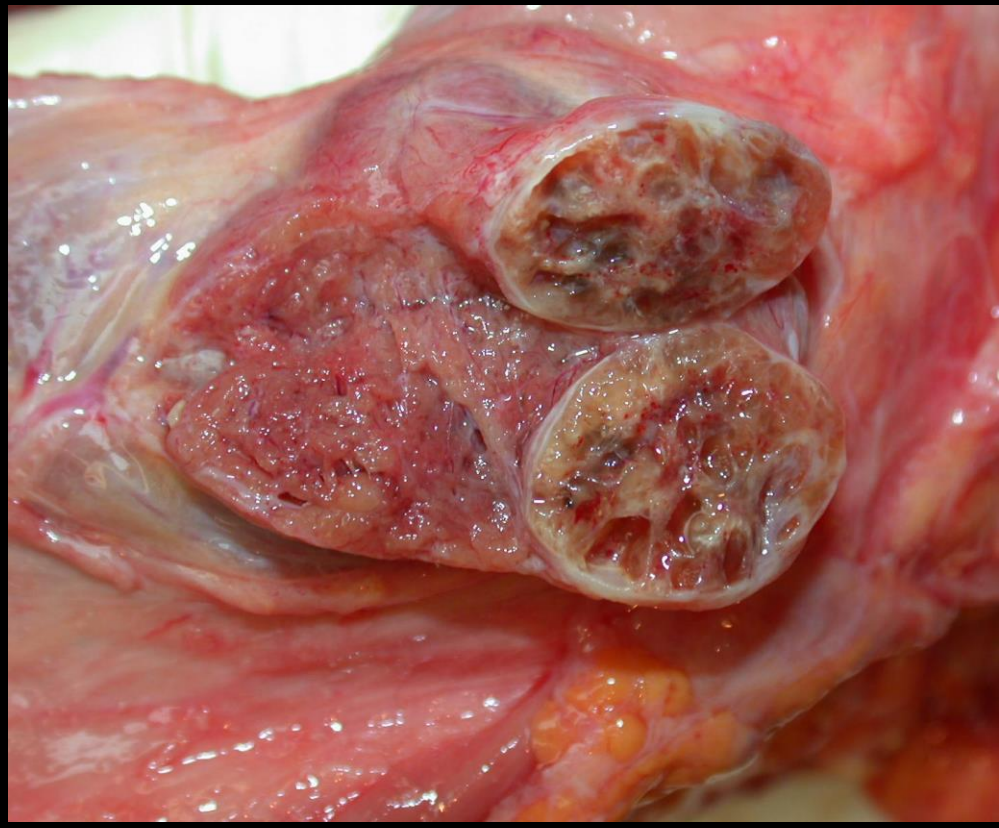
when one nodule becomes autonomous




4.2
11L5
T8.4
CF 7.2
13 fps
2DG
99
DR
60
CG
40
PRF
10.9k
Filter
4
11L5
T8.4
16 fps


MI:0.9
2DG
99
DR
60





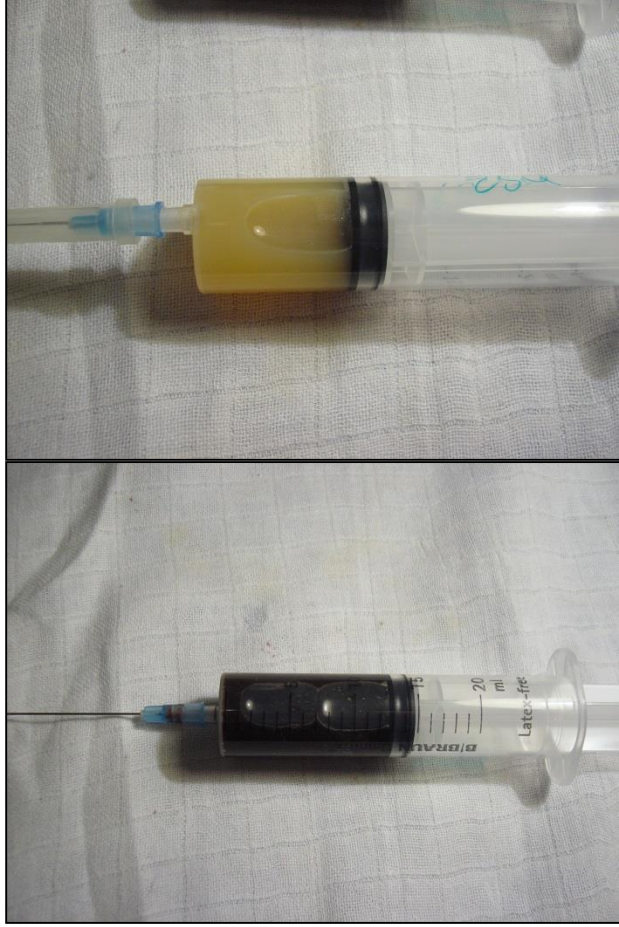
Thyroid cyst

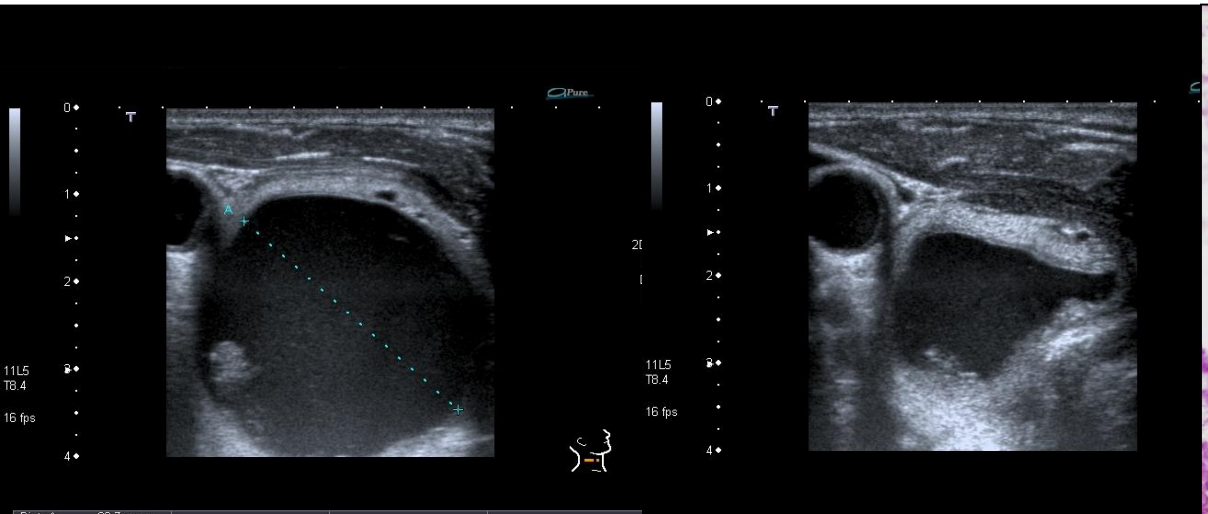
Might be „cold"

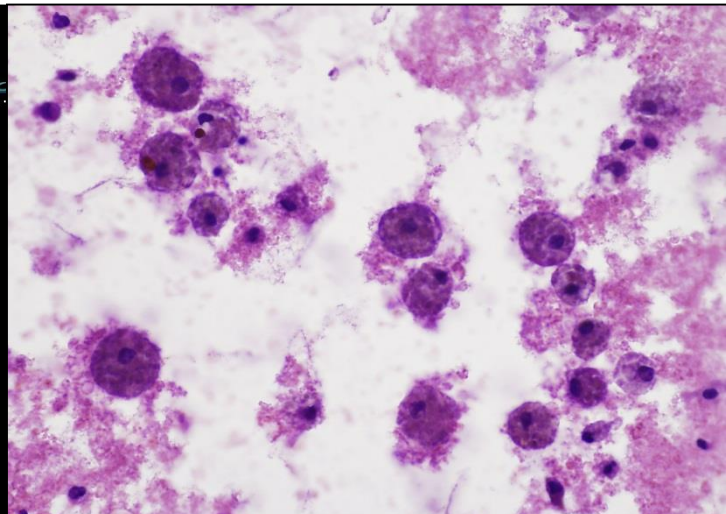
or „warm" nodule

Scintigraphy frequently consideres it

„autonomous adenoma"







Tumors

Suspicious, if:

Solitary nodule
Young patient
Male
Cold (I, ! , Tc might show it to be hot !)

Tumors

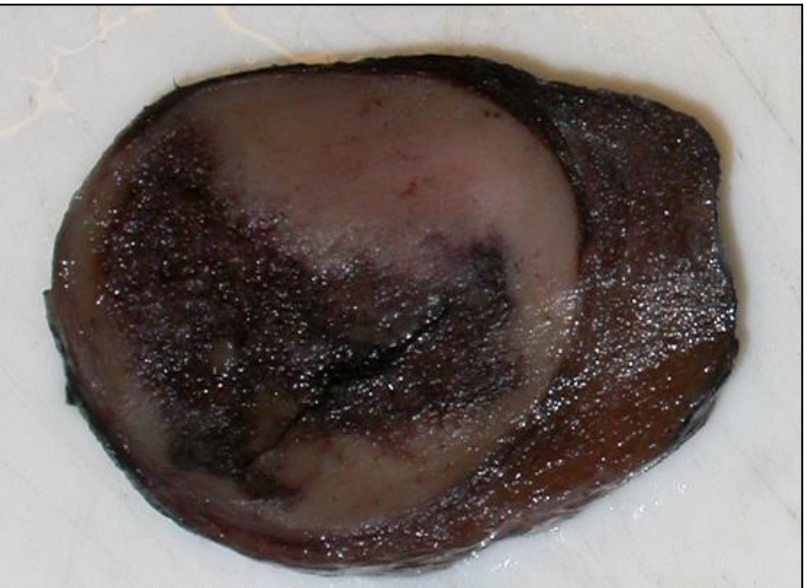
Adenoma

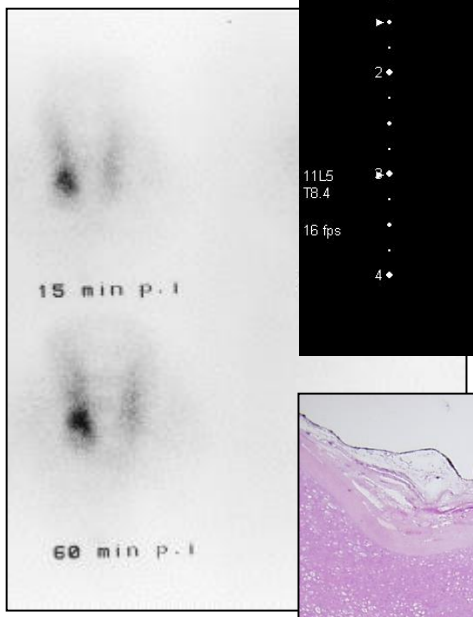
Non-functioning

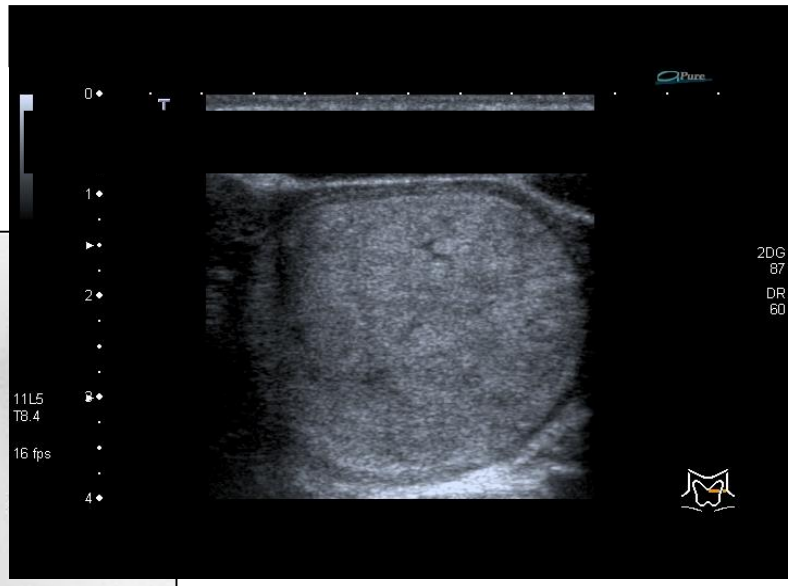
(frequently „cold")

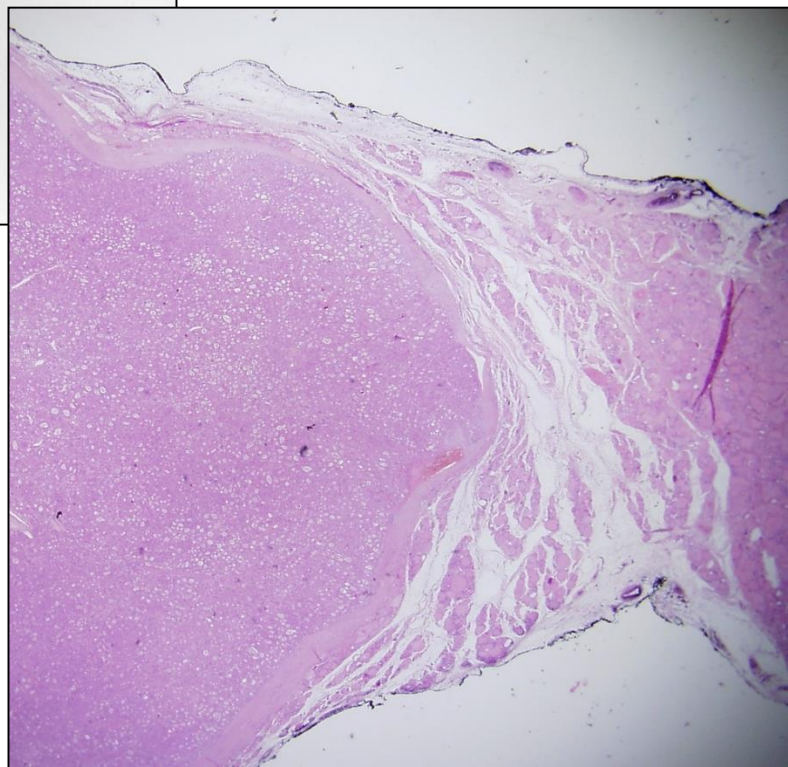
Hormon-producing

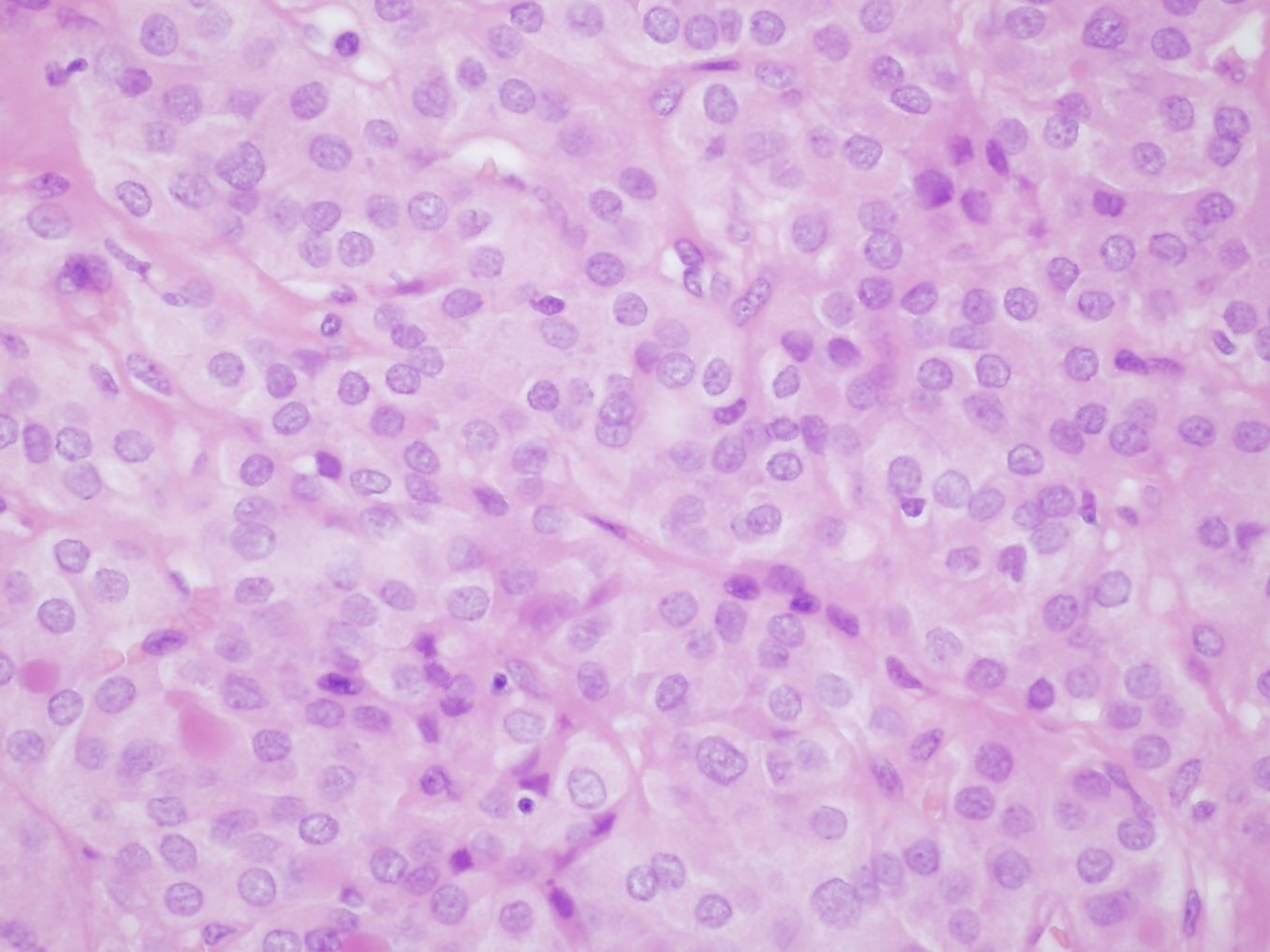
(„warm", „hot nodule"
toxic adenoma)

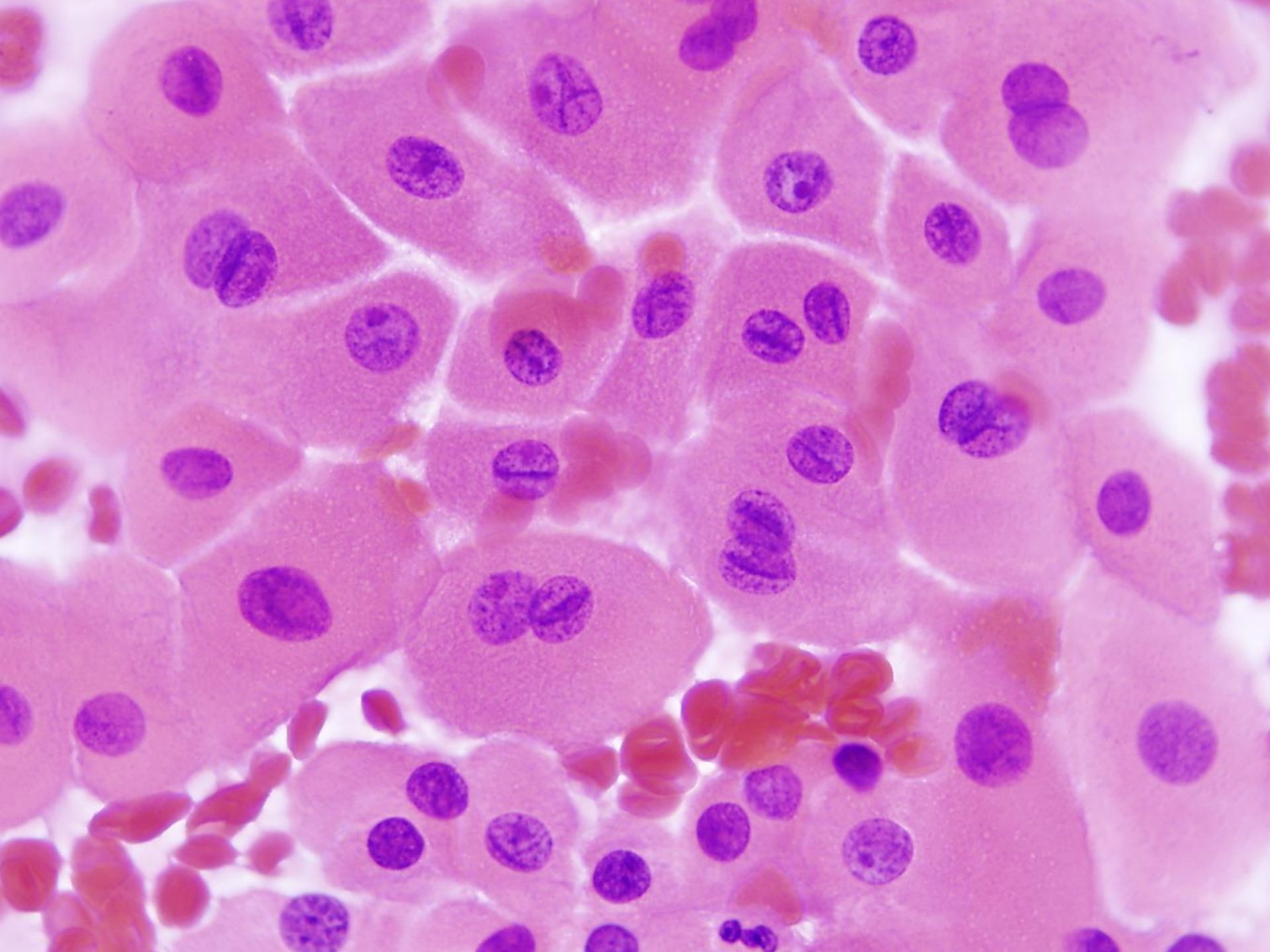












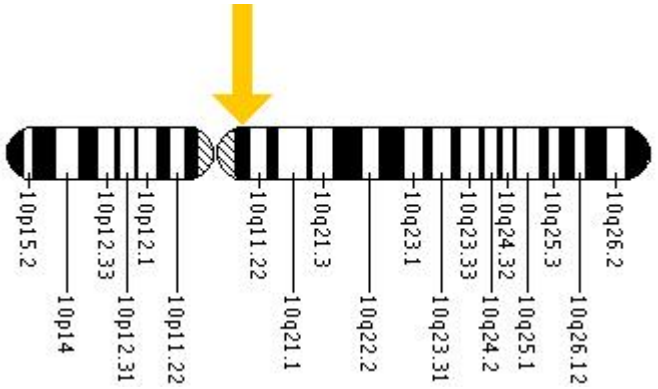
Relative frequency of malignant thyroid tumors

- Papillary carcinoma	75%
- Follicular carcinoma	15%
- Medullarycarcinoma	5%
- Anaplastic cc.	2%
- Lymphoma	2%
- Other, non-epithelial	0.8%
- Metastatic	0.2%

Malignant thyroid tumors

Genetic background – mutations

ex.: ret/PTC



Ionizing irradiation

(therapeutic,

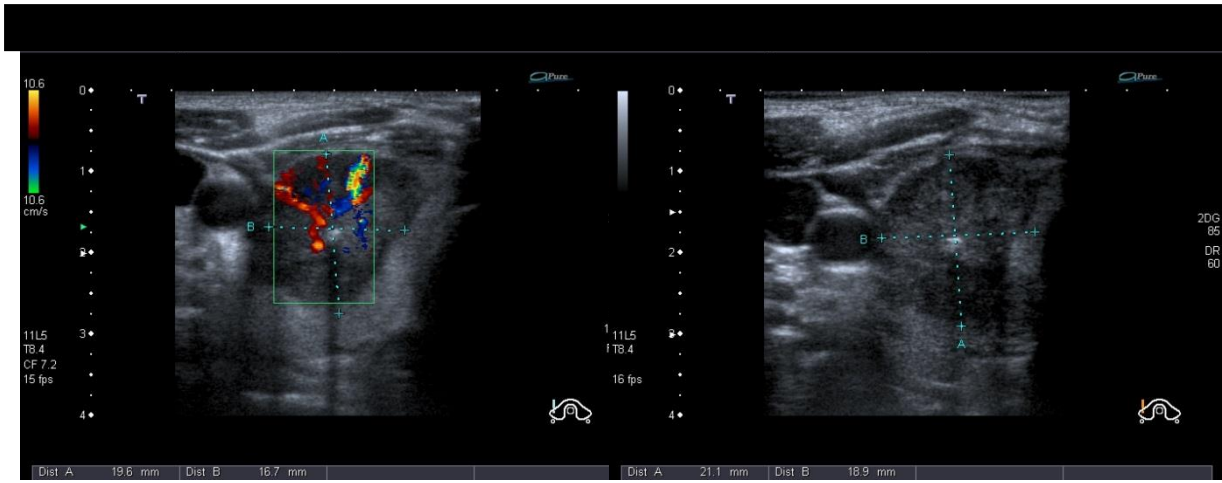
environmental)

Papillary cc.

Occurrence: middle aged women, any age, males can be affected

Signs

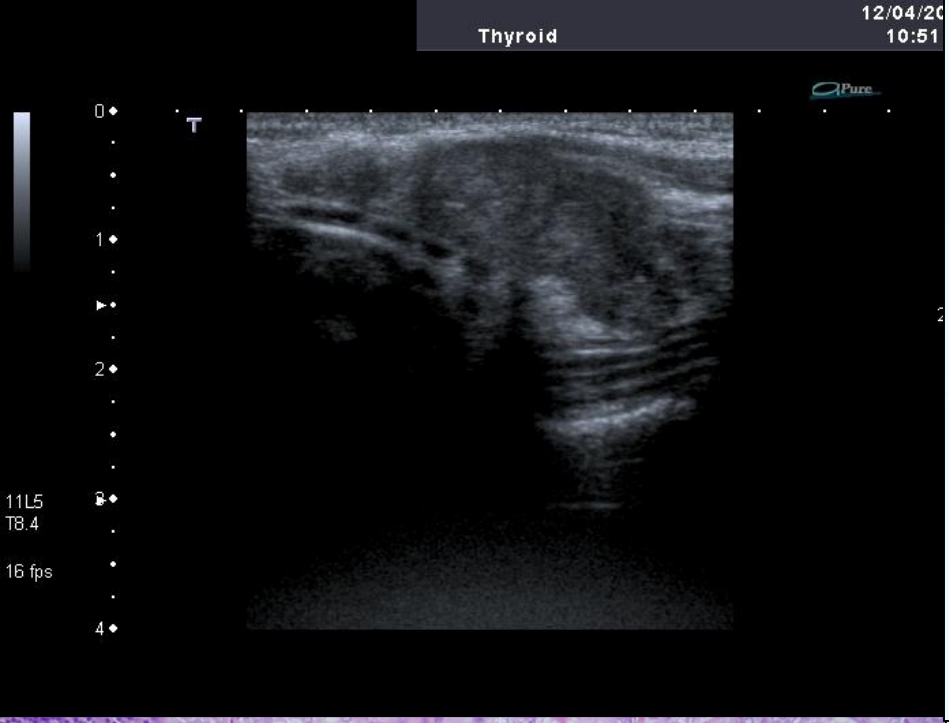
- „Nodule"
- Hoarseness
- Cough
- Dyspnoe

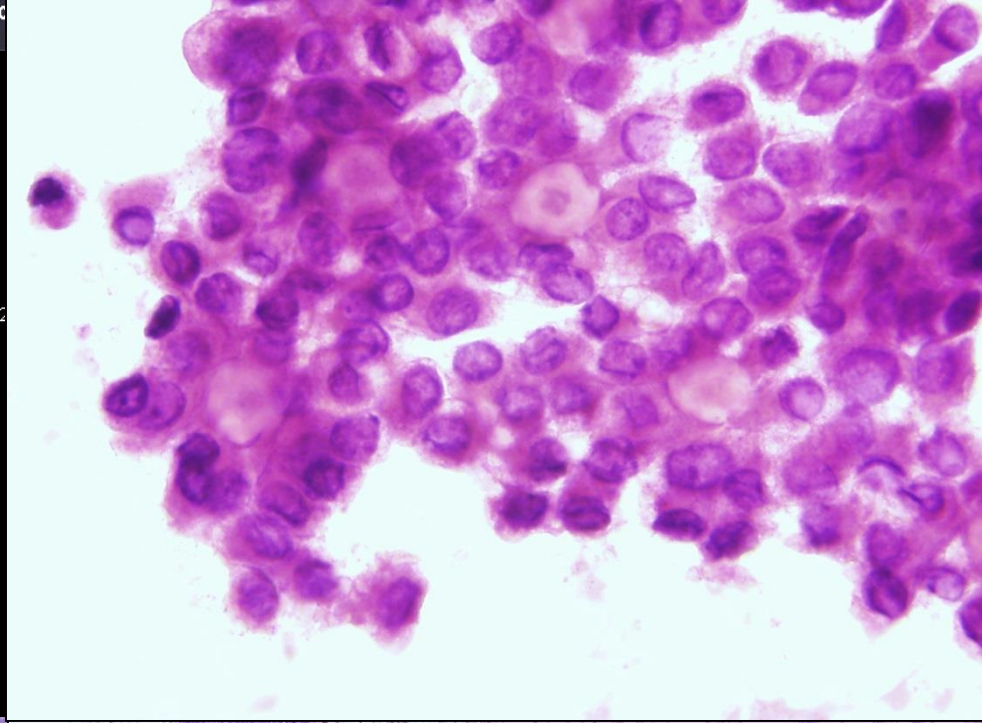


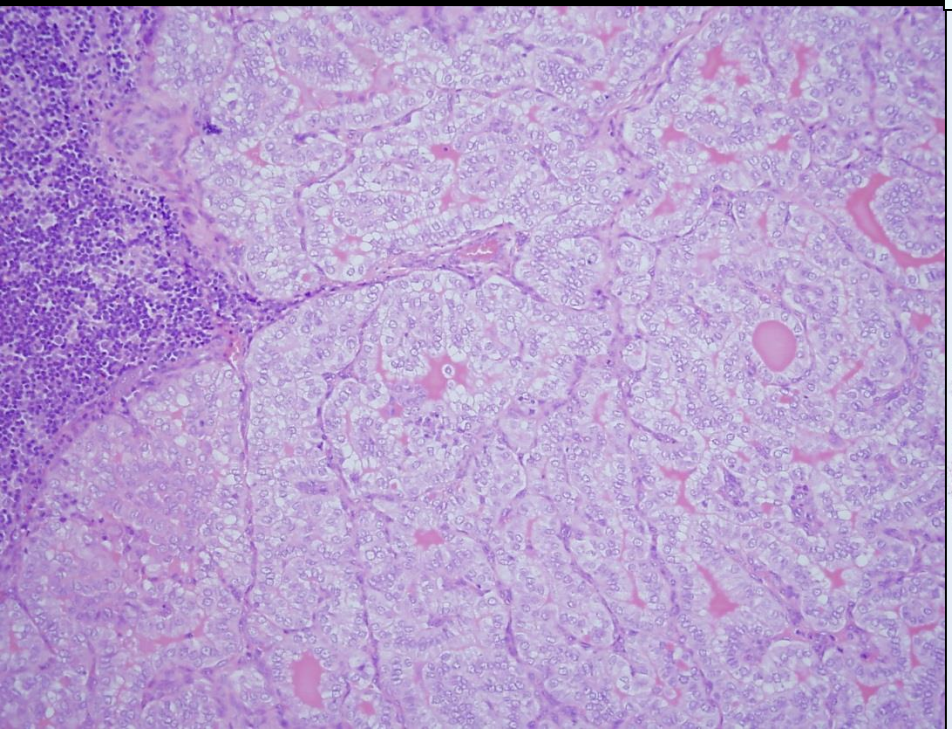
Metastasis: regional ln-s, ***rarely*** distant

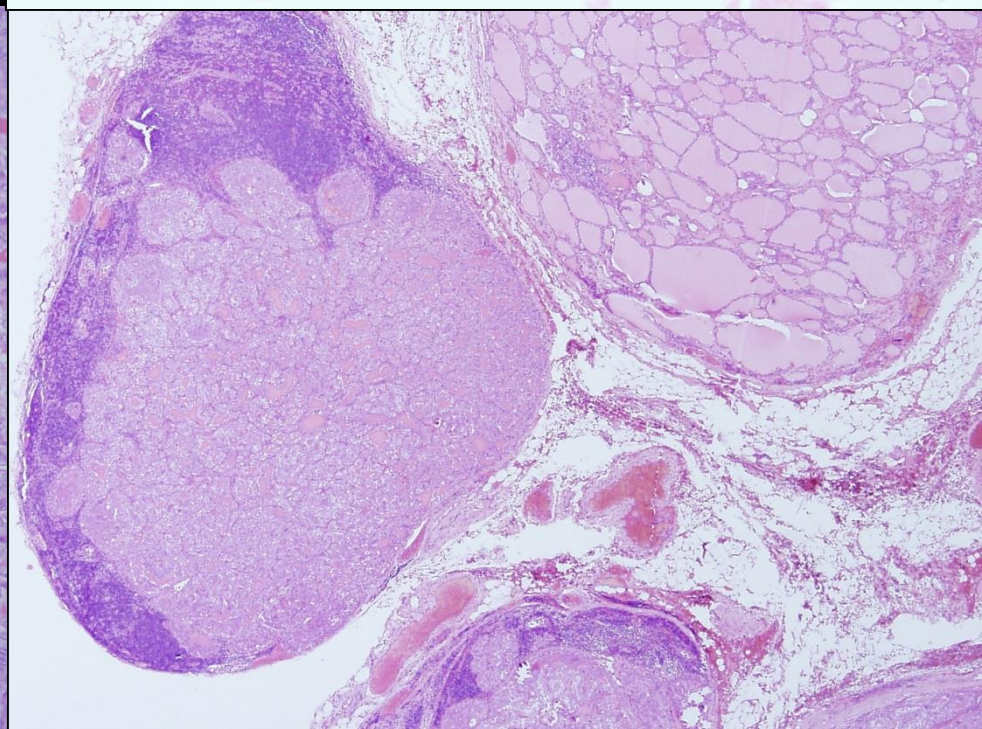
Prognosis: relatively good

Th.: surgical + radioiodine th.


Thyroid
12/04/20
10:51
T
11L5
T8.4
16 fps





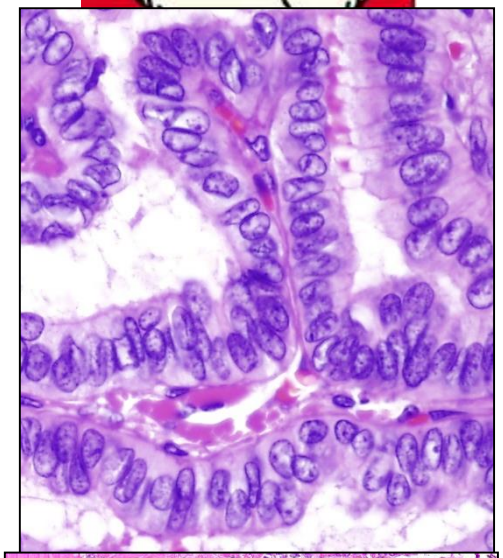


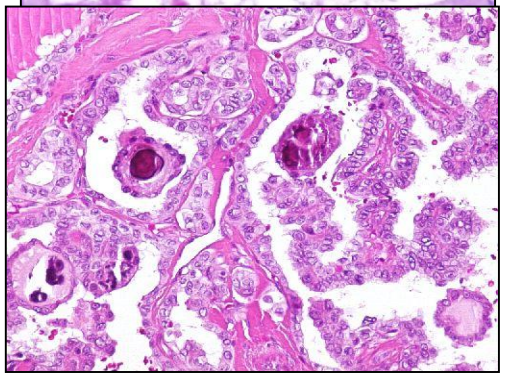
Papillary cc.

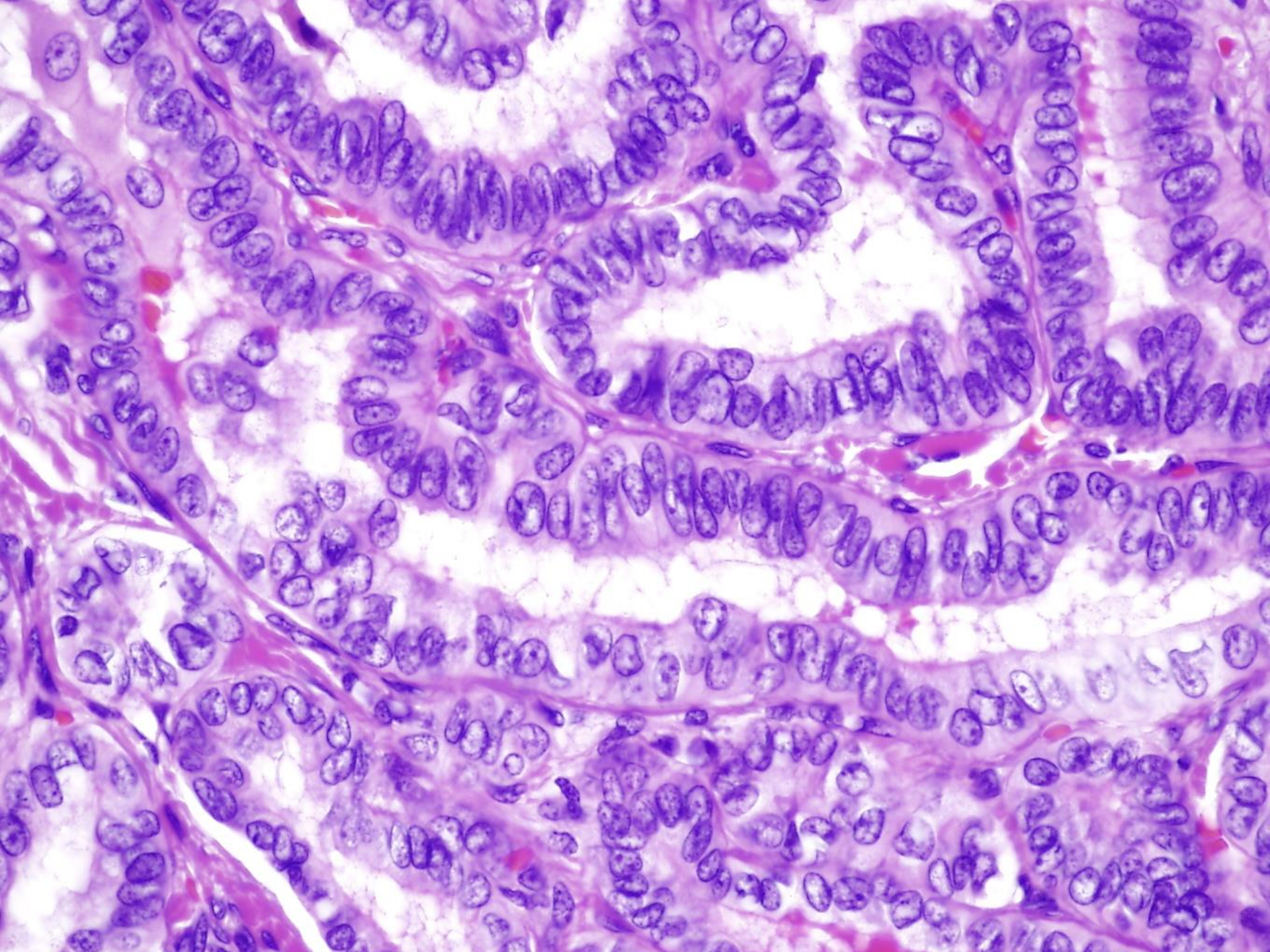
„Classic" (papillae, Orphan Annie, Psammoma)

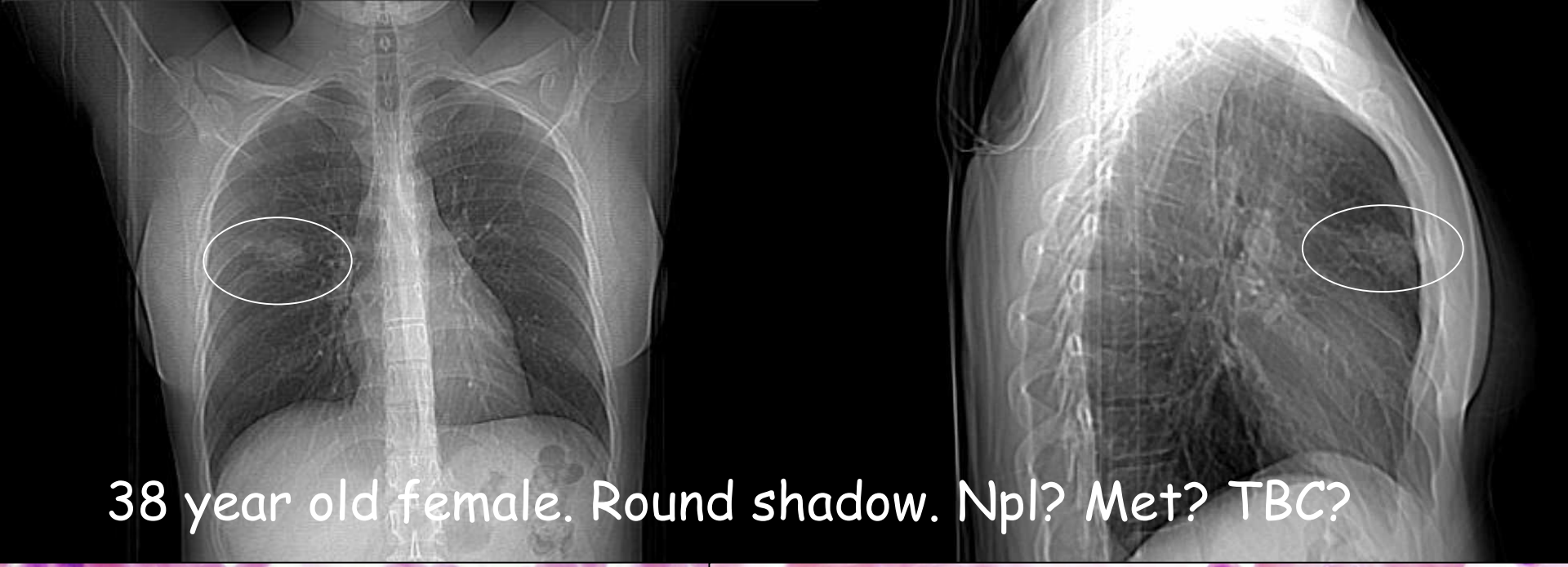
Specific types

- Encapsulted
- Follicular
- Tall cell
- Diffuse sclerotizing (children)
- Hyalinizing trabecular

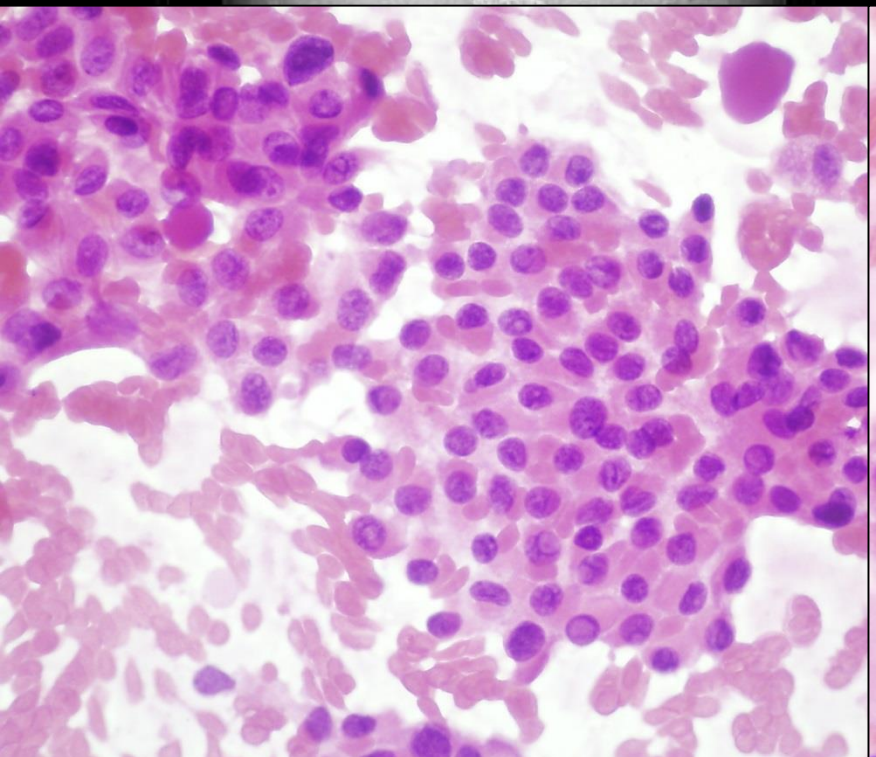


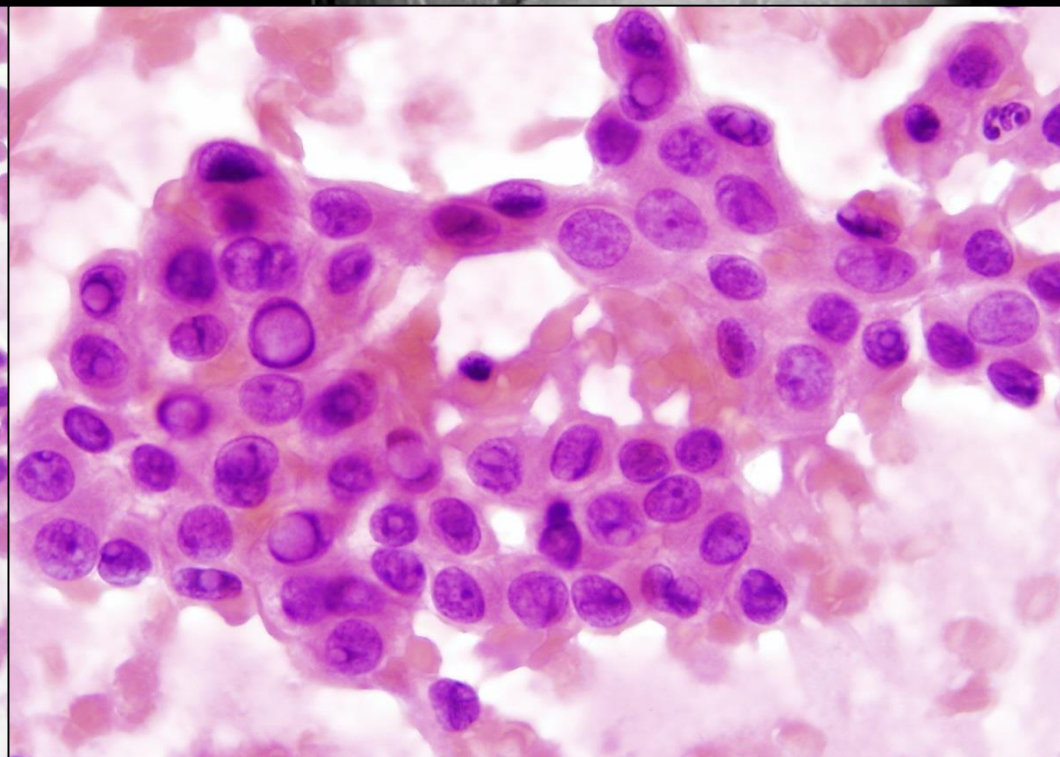


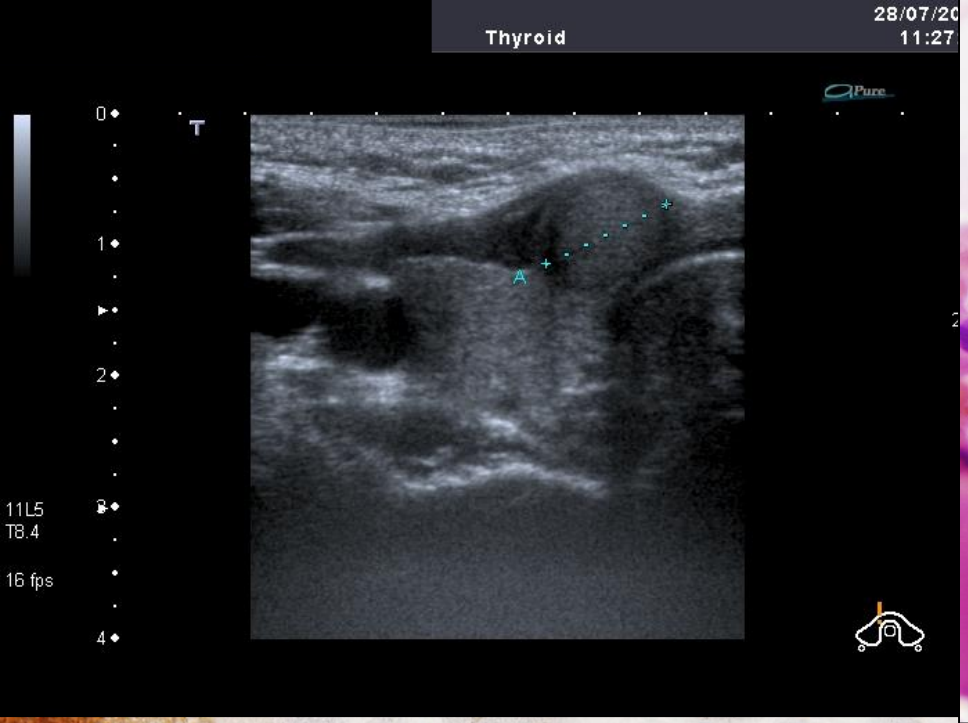


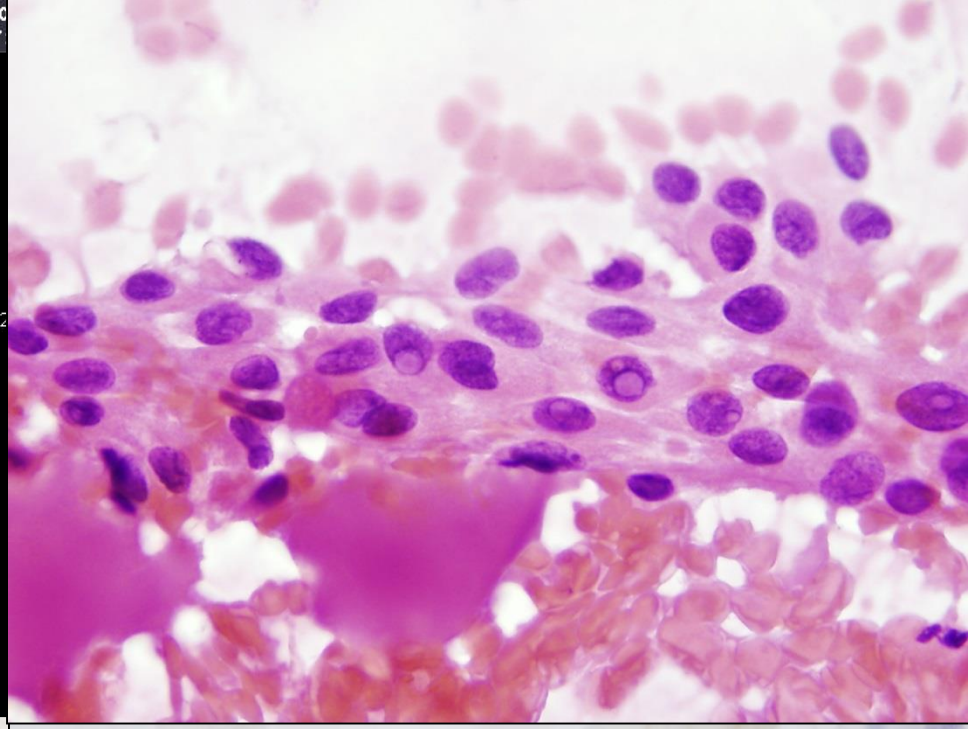


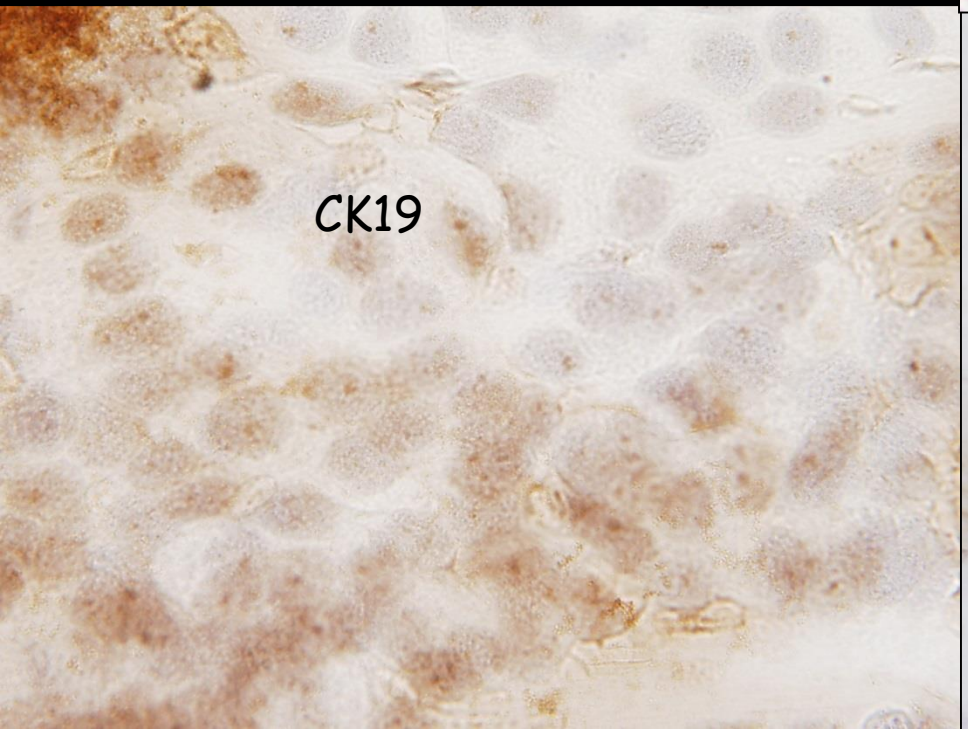
38 year old female. Round shadow. Npl? Met? TBC?

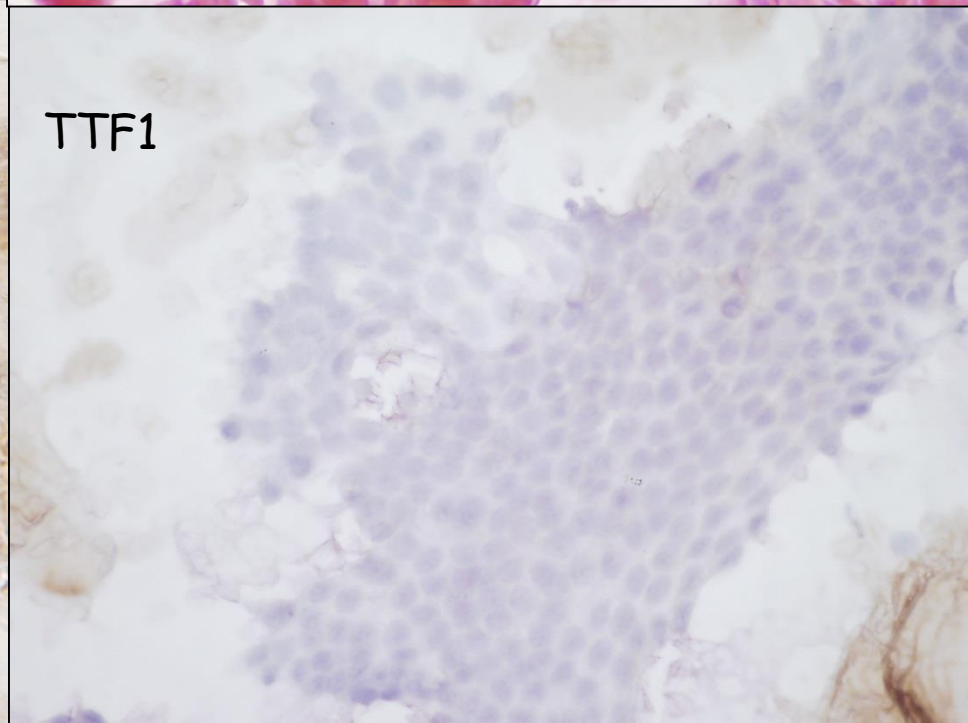


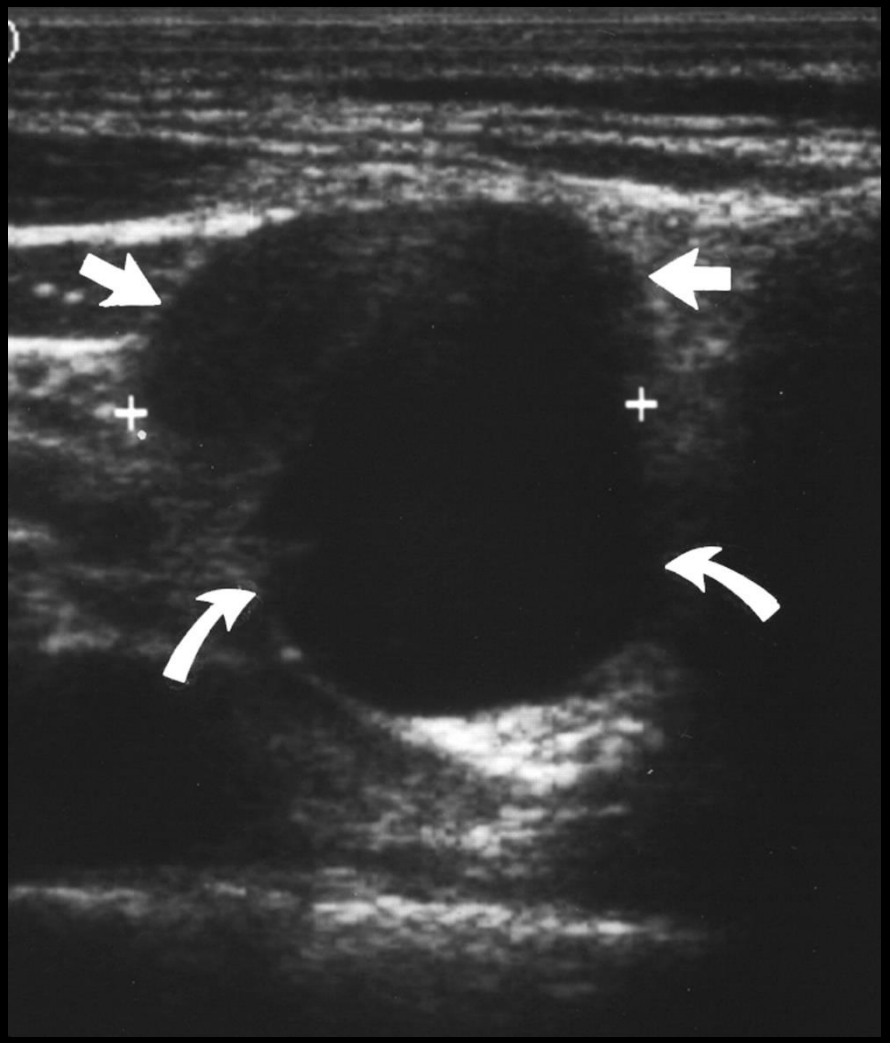


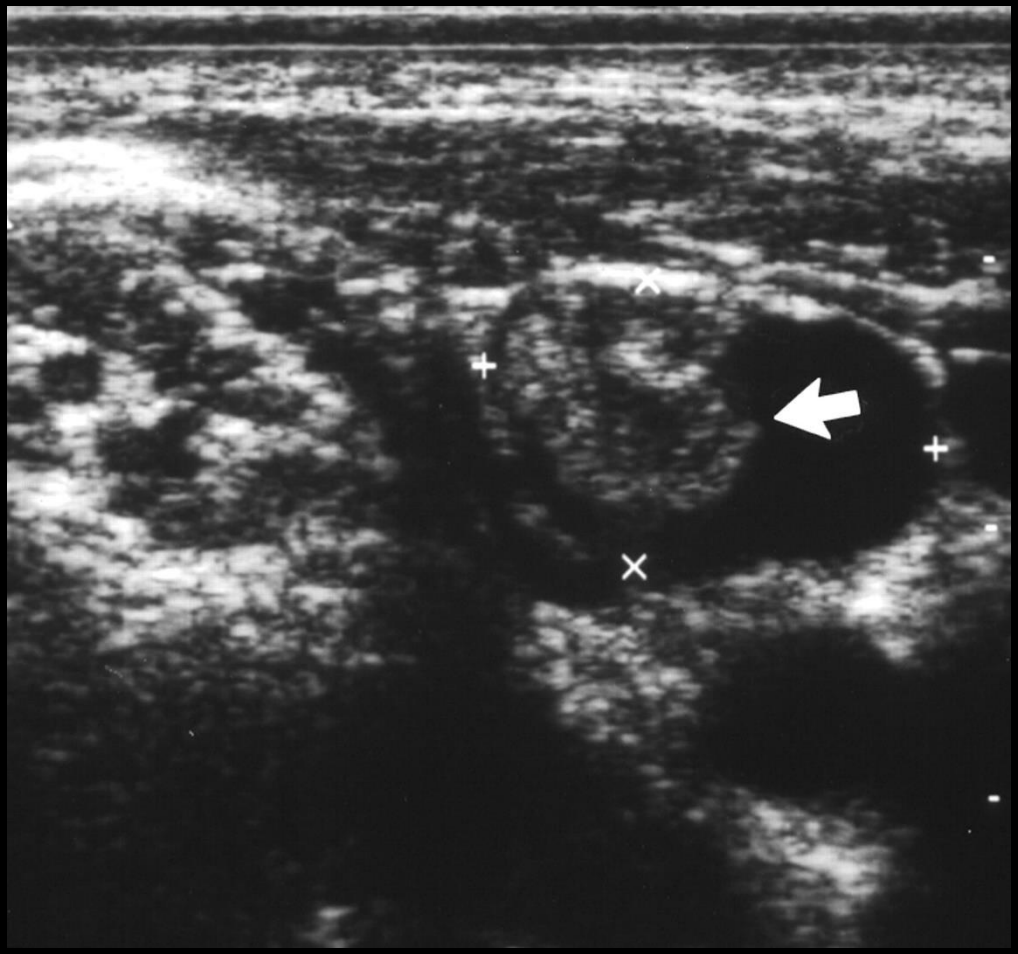

Thyroid
28/07/20
11:27
11L5
T8.4
16 fps

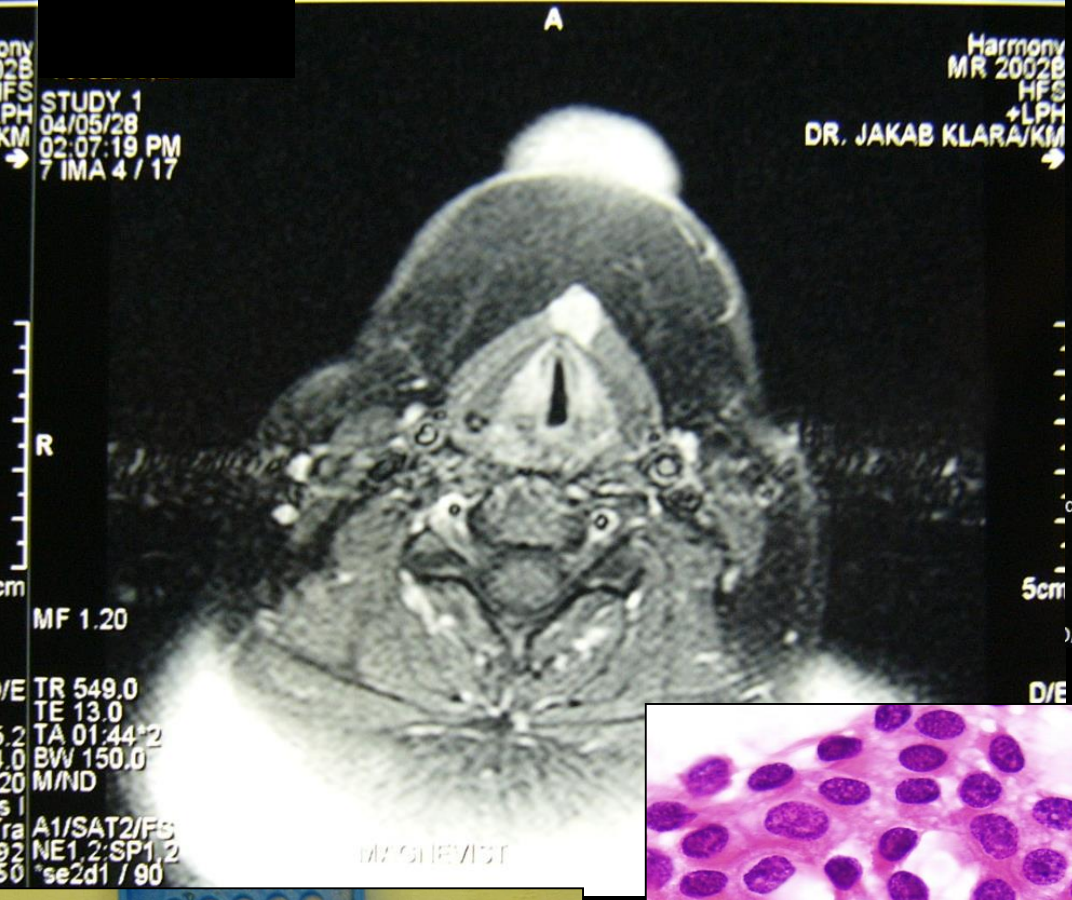


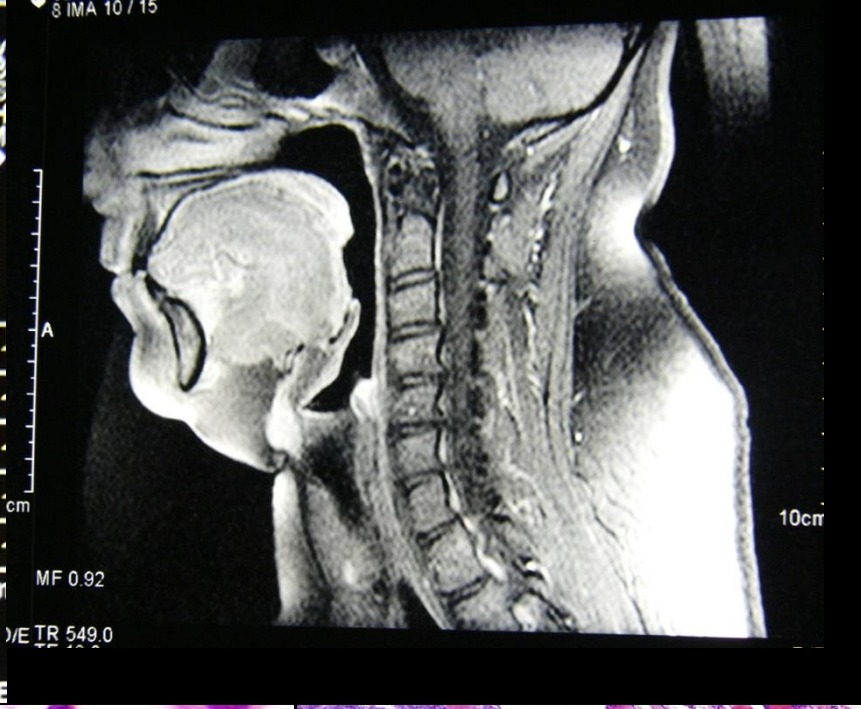

CK19

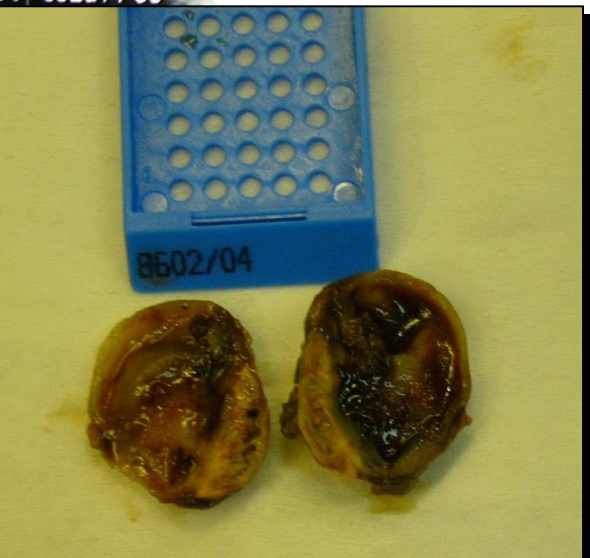

TTF1

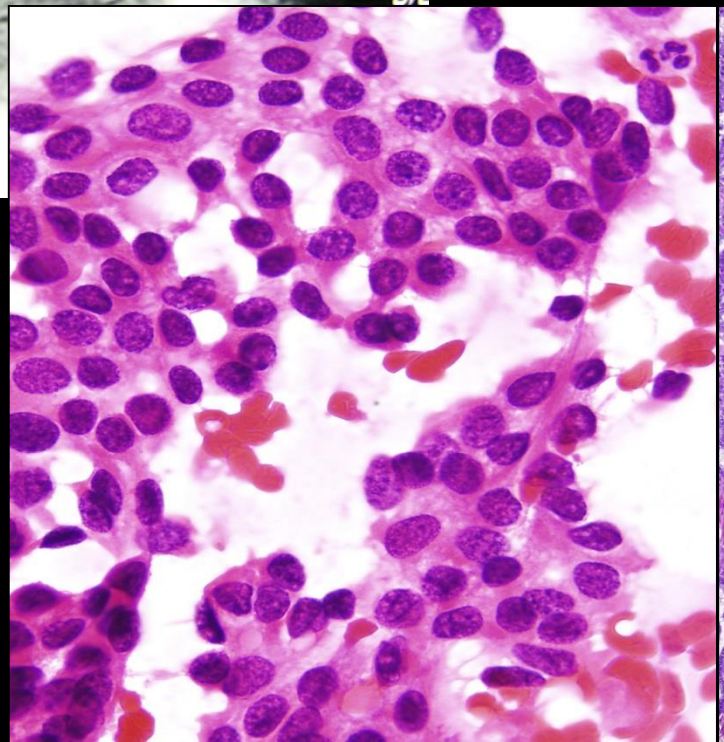


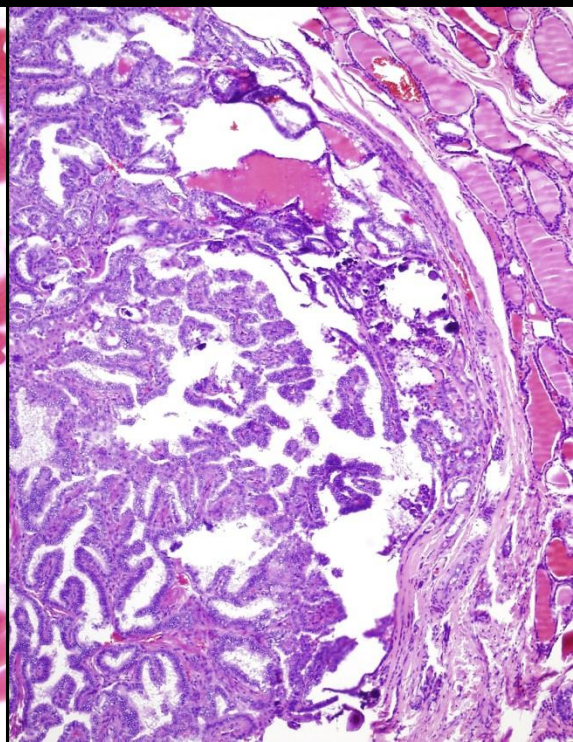



A
Harmony
MR 2002B
HFS
+LPH
DR. JAKAB KLARA/KM
STUDY 1
04/05/28
02:07:19 PM
7 IMA 4 / 17
R
5cm
MF 1.20
TR 549.0
TE 13.0
TA 01:44*2
BW 150.0
M/ND
A1/SAT2/FS
NE1.2:SP1.2
*se2d1 / 90


8 IMA 10 / 15
A
cm
10cm
MF 0.92
TR 549.0


8602/04





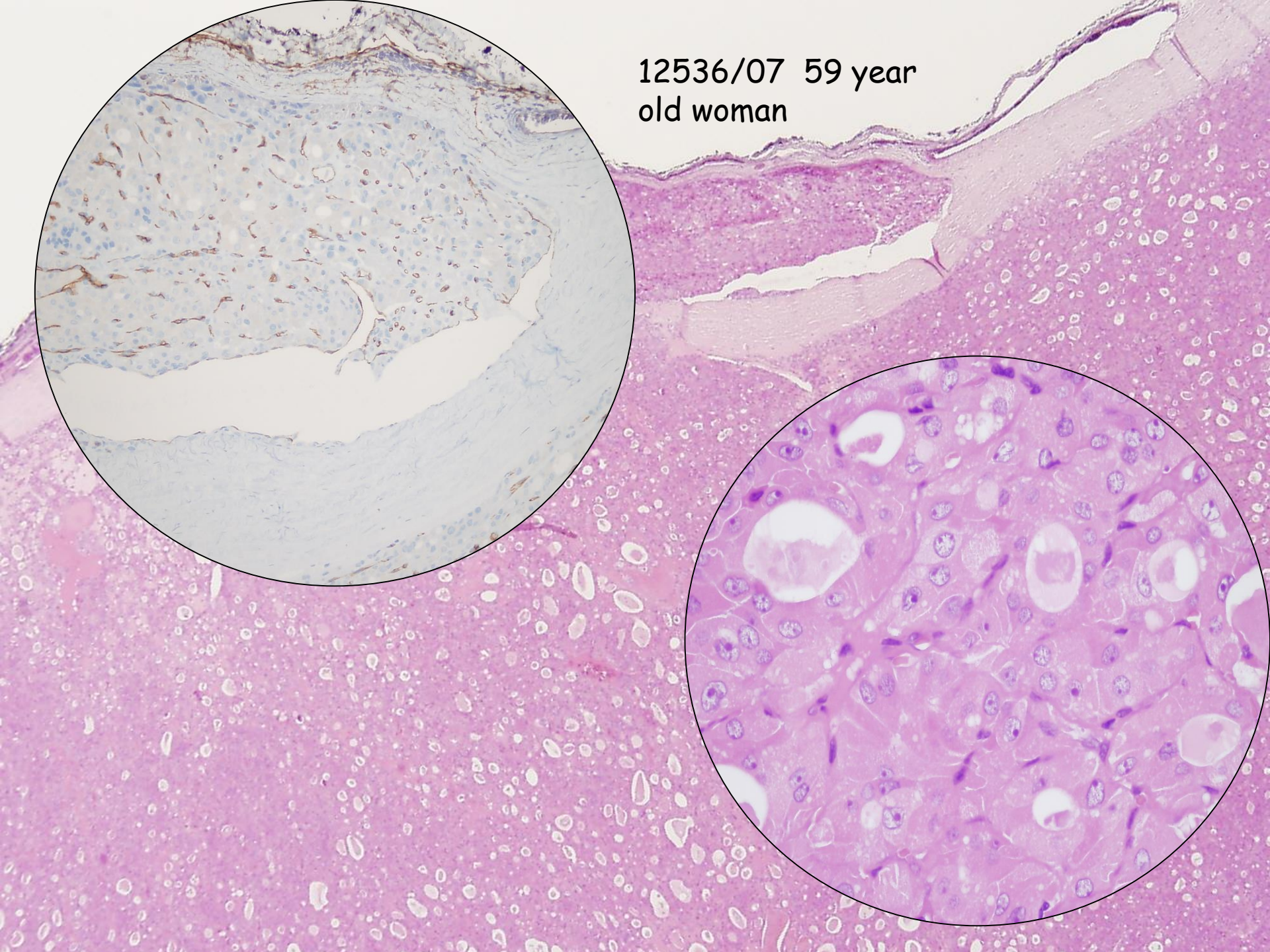
Follicular cc.

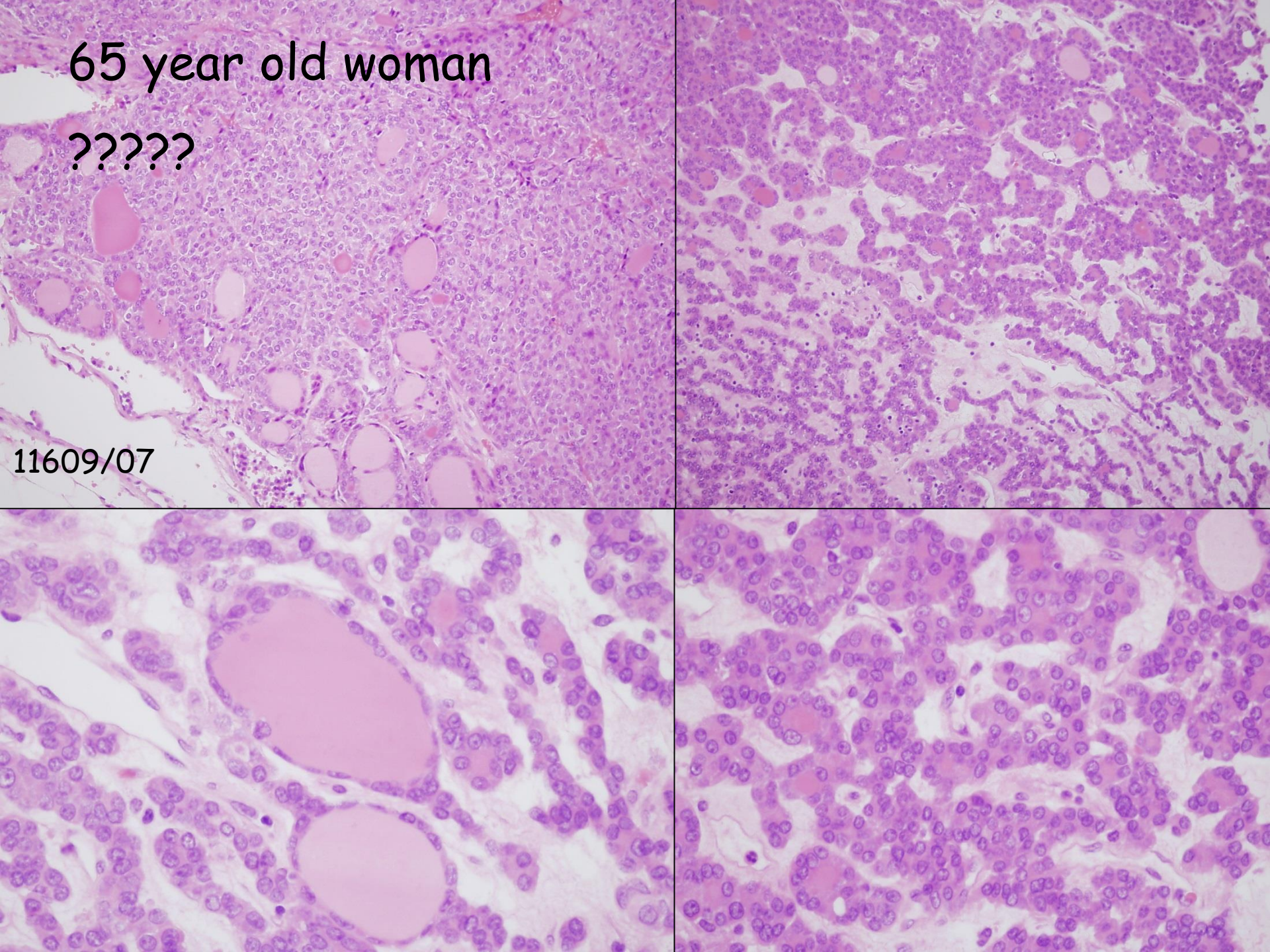
Occurrence
  Elder women

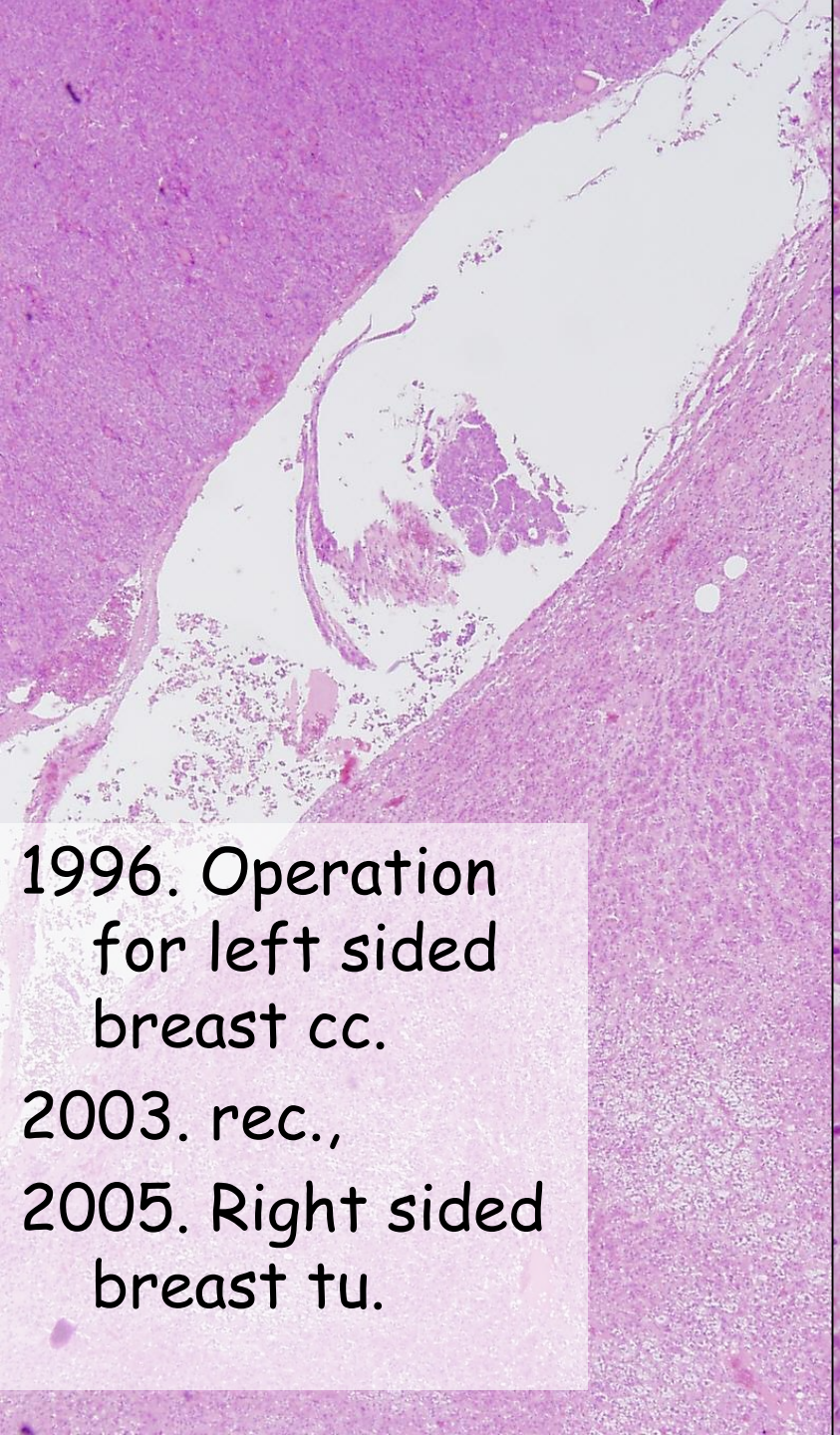
Cause: ras mutation (in foll. Adenomas also)
Slowly growing nodule (usually cold, rarely warm)

Monotonous cells
Capsule / and/ or vascular invasion
Reg. Lymph nodes are rarely metastatic, but
  liver, bones are frequently metastatic sites

Progn.: Depends on the metastatic capacity
Th.: surgical + radioiodine th


12536/07 59 year
old woman

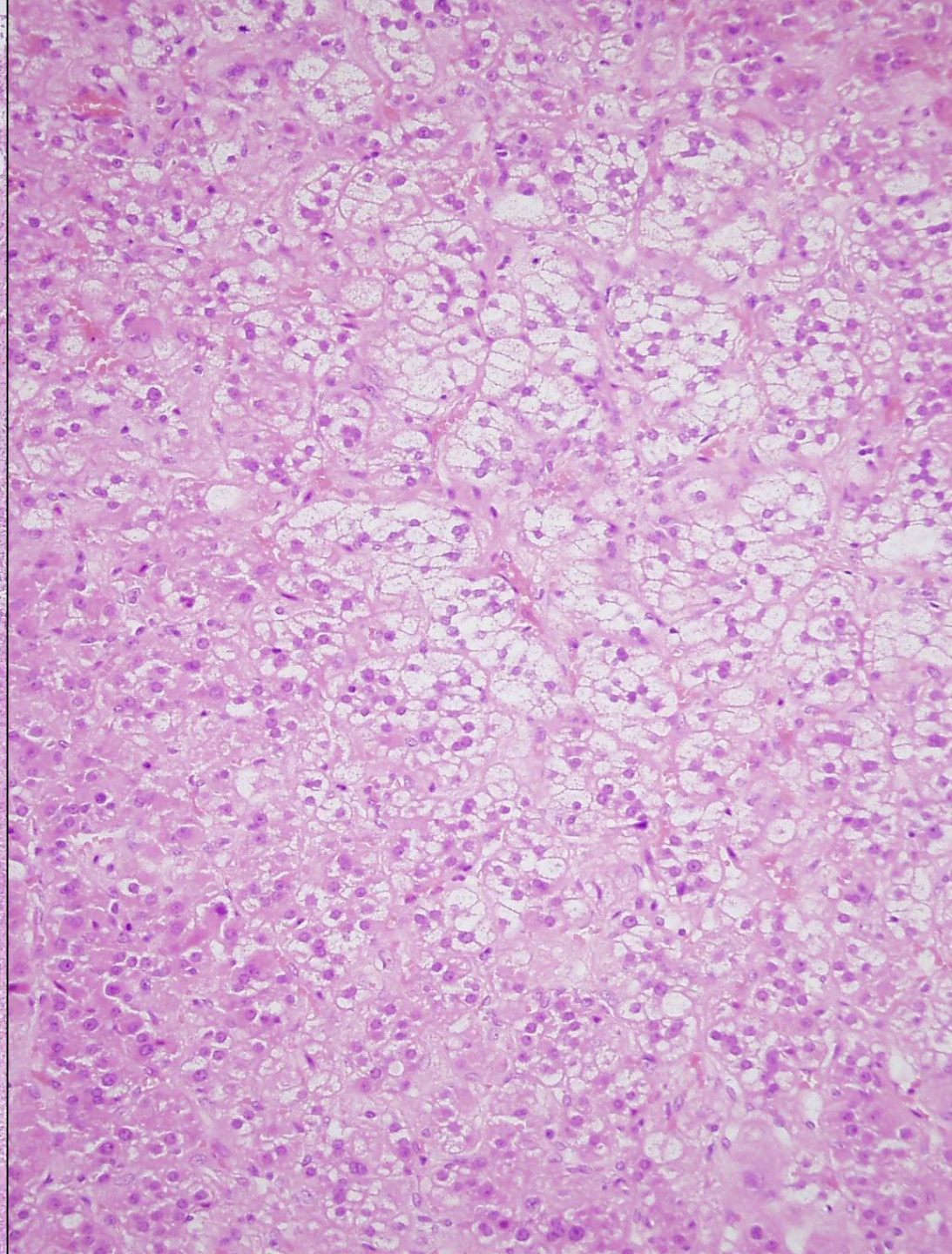

65 year old woman
?????
11609/07

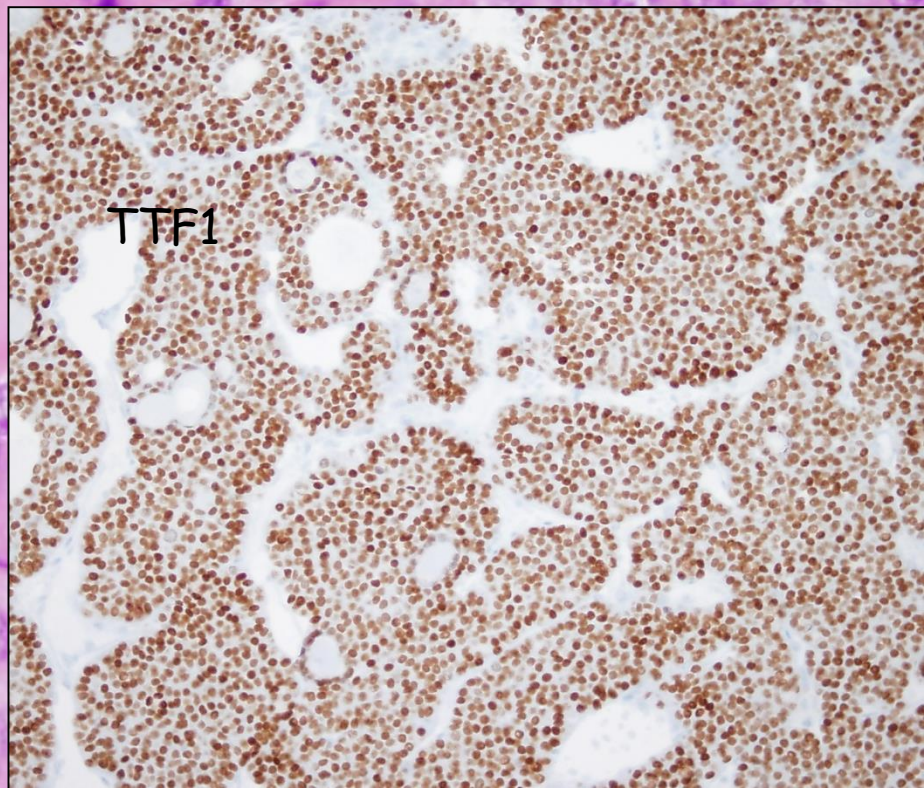


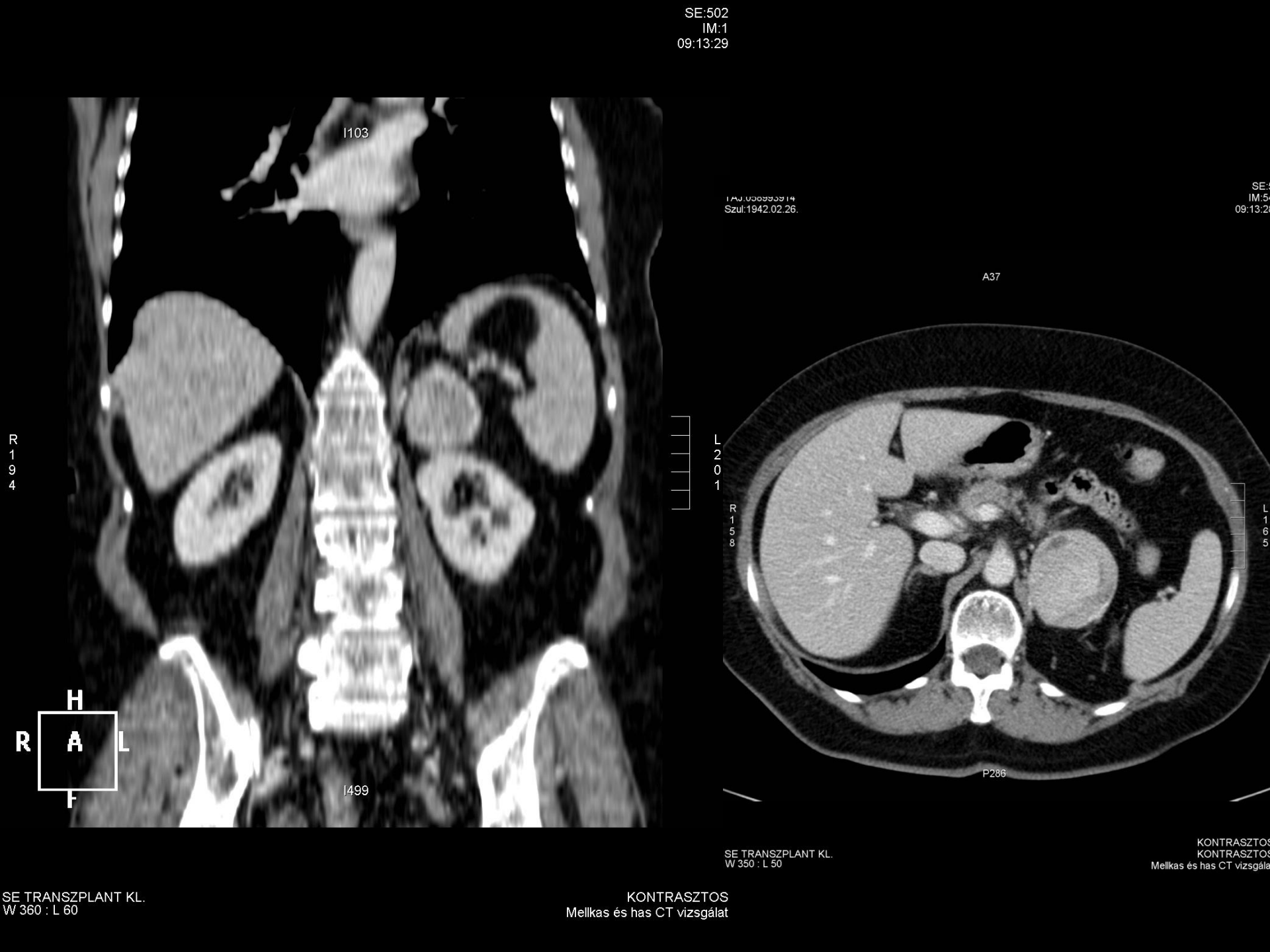
1996. Operation for left sided breast cc.

2003. rec.,

2005. Right sided breast tu.



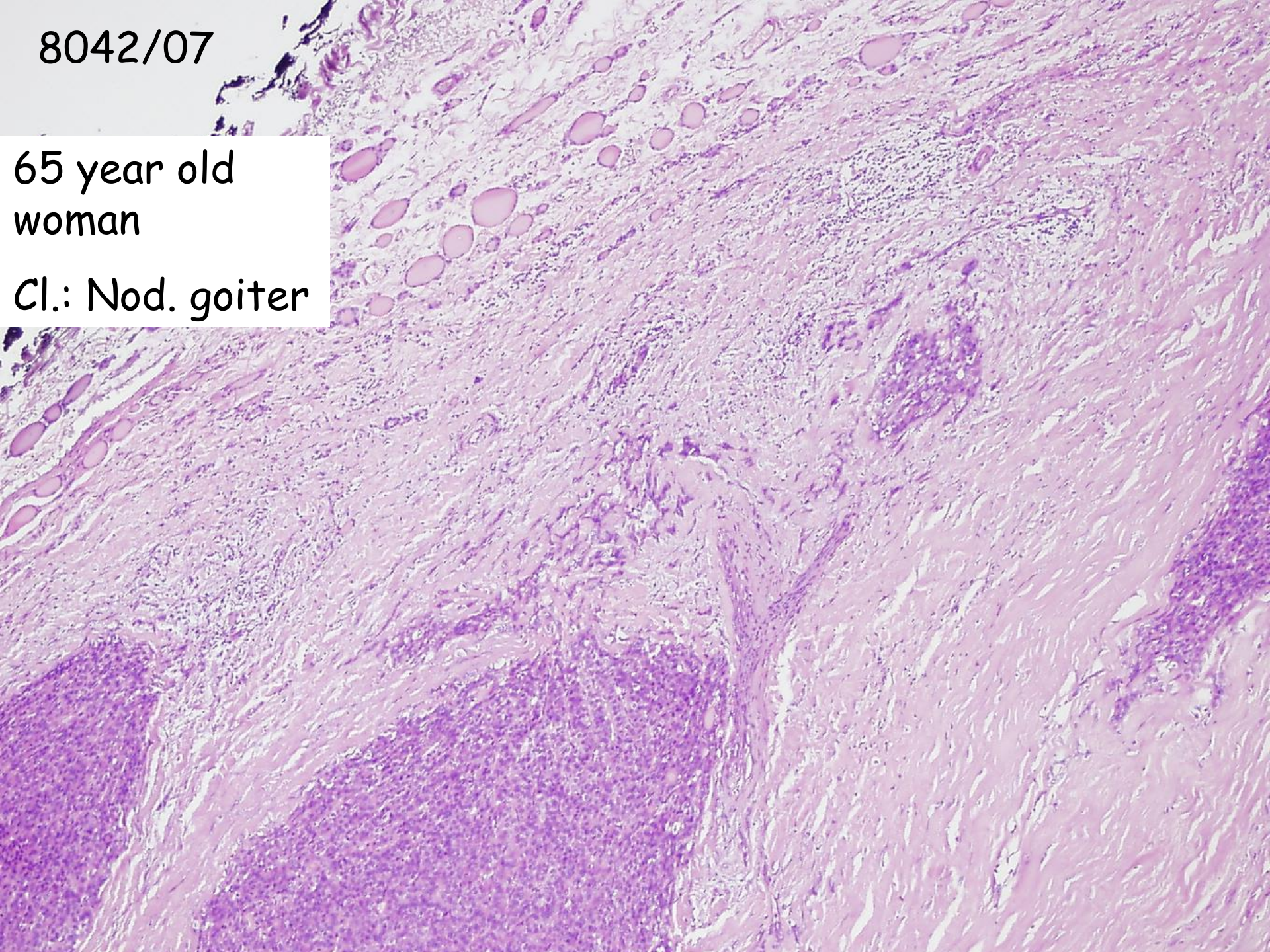

TTF1

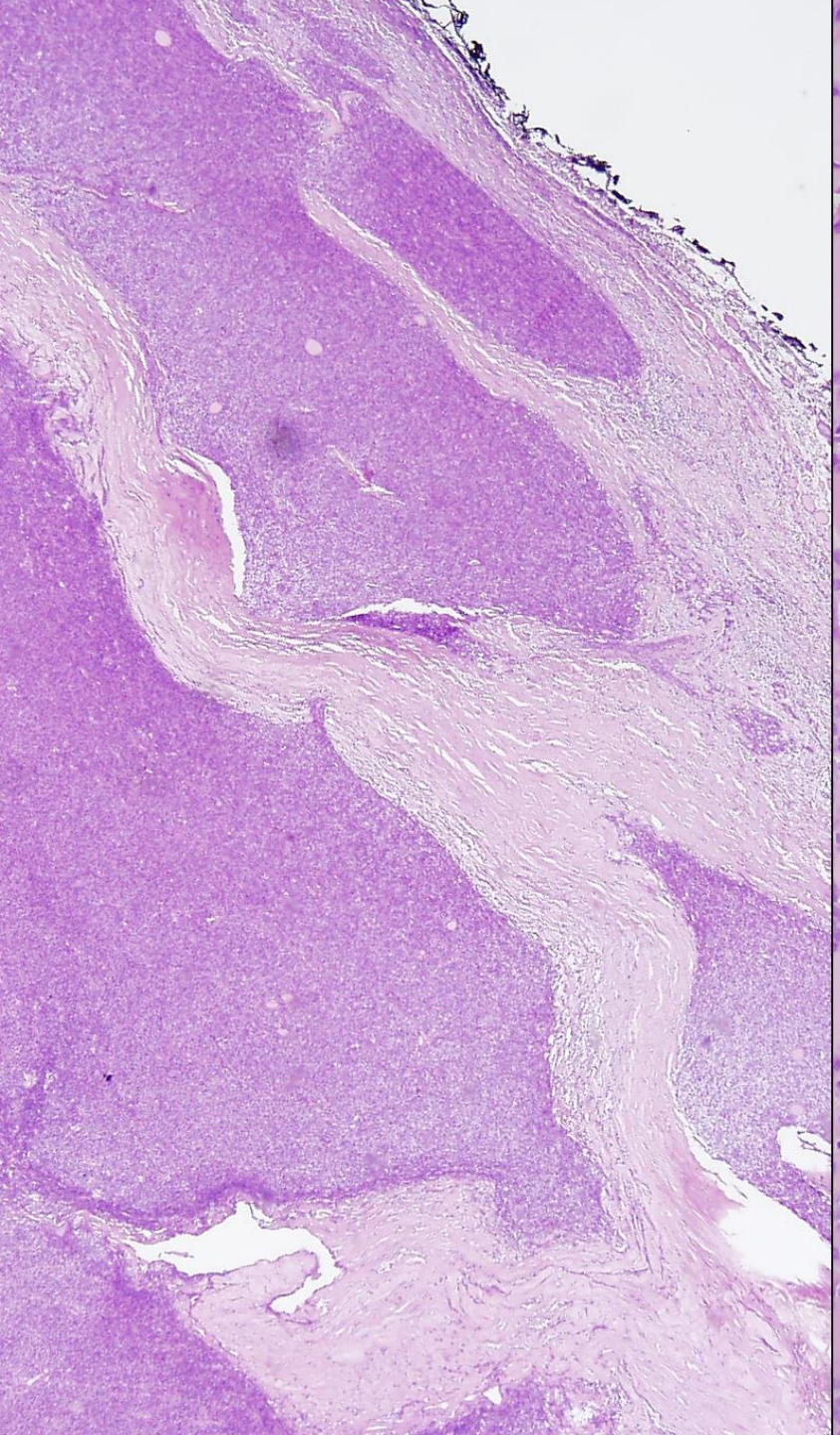

SE:502
IM:1
09:13:29
I103
R
1
9
4
H
R
A
L
F
I499
Szul:1942.02.26.
A37
L
2
0
1
R
1
5
8
P286
SE TRANSZPLANT KL.
W 350 : L 50
KONTRASZTOS
SE TRANSZPLANT KL.
W 360 : L 60
KONTRASZTOS
Mellkas és has CT vizsgálat

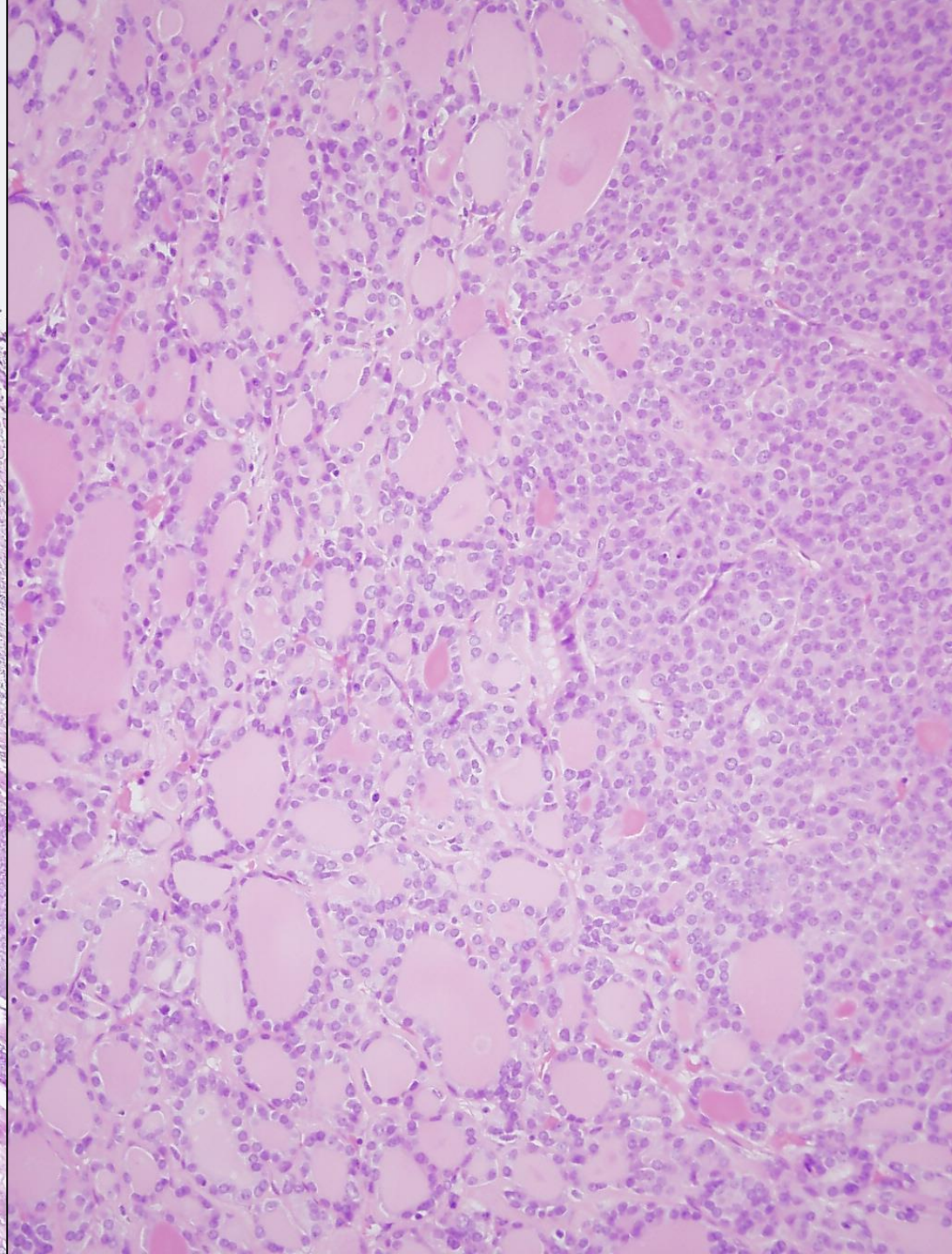
8042/07

65 year old woman

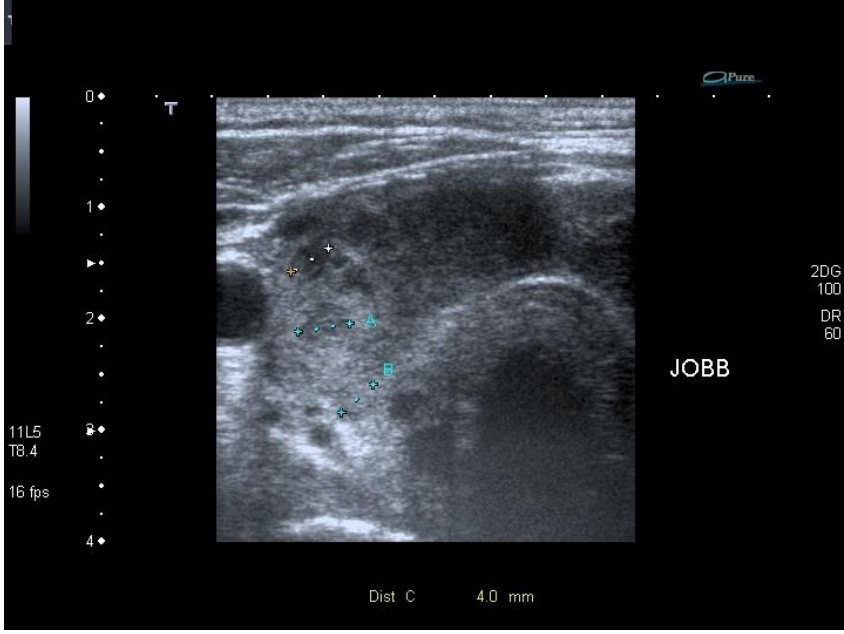
Cl.: Nod. goiter







Anaplastic cc.

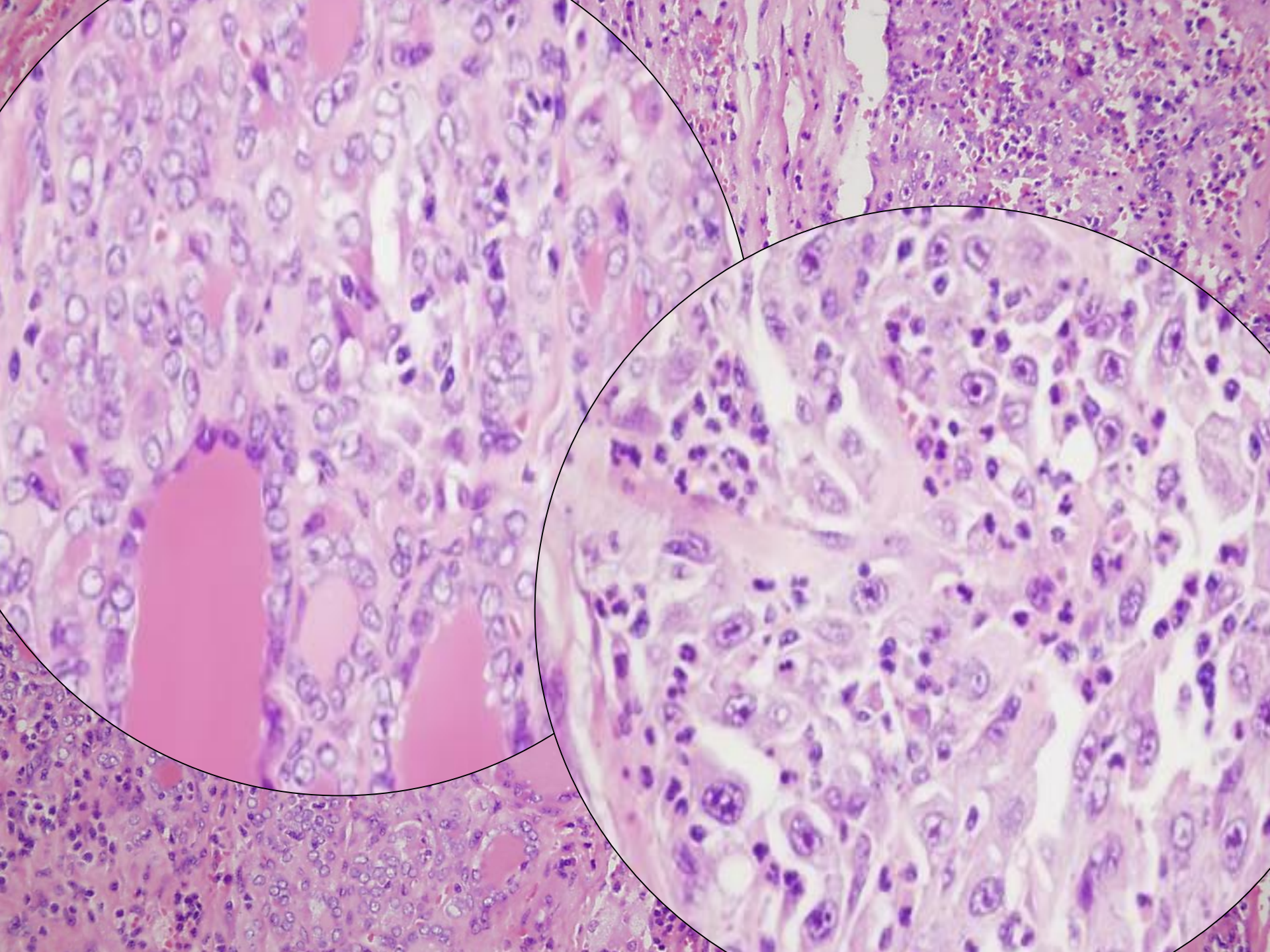


Based on some kind of thyroid disease
(nodular goiter, or papillary thyroid cc.)

Rapidly growing, agressive tumor.

Hoarseness, suffocation, compression

Mortality: 100 %, in one year



Medullary cc.

C cells

80 % sporadic

20 % a MEN sy 2A, 2B. or

FMTC

(Familiary medullary thyroid cc. FMTC,- spec. MEN2A)

Solitary nodule (sporadic), or multiple smaller familiary (on the basis of C cell hpl)

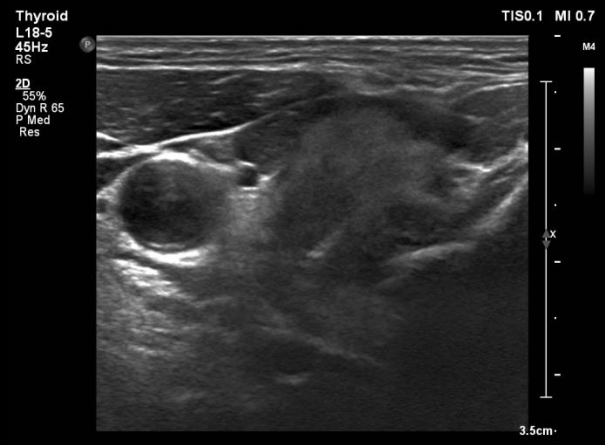
Medullary cc.

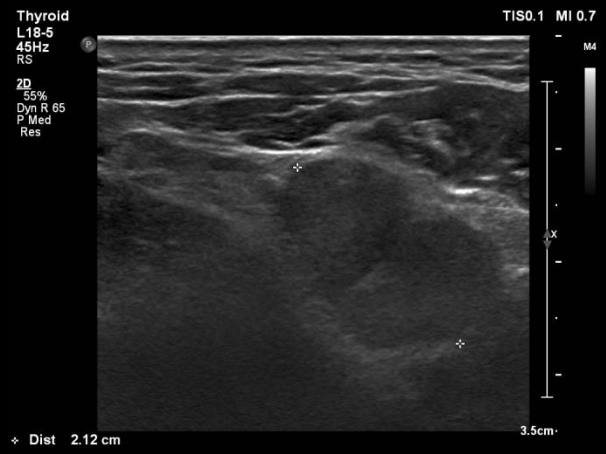
Symptoms: nodule, horseness, dysphagy
paraneoplastic (?!) hormone production

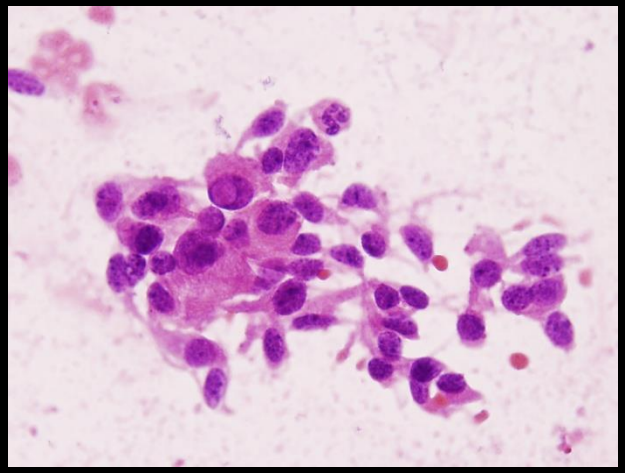
Calcitonin ^^, but hypocalcaemia cannot be always shown

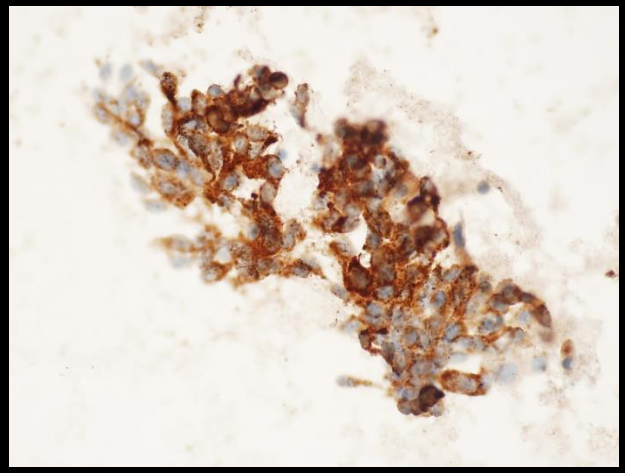
Familiary cases: RET-mutation is found in case of family screening. (C cell hpl might be found in prophilactically resected thyroids)

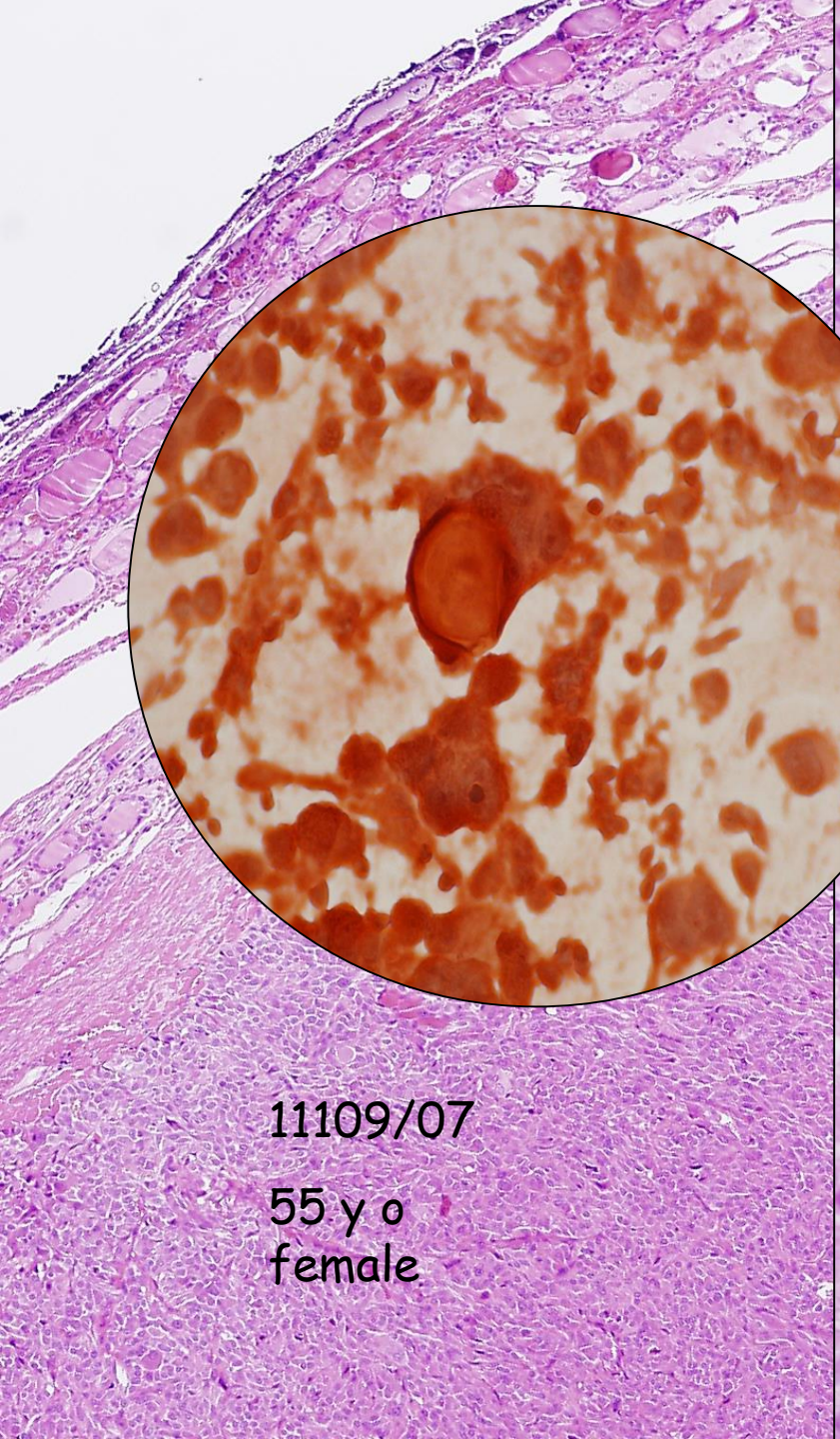
Male, 53
Examination for swelling of the neck





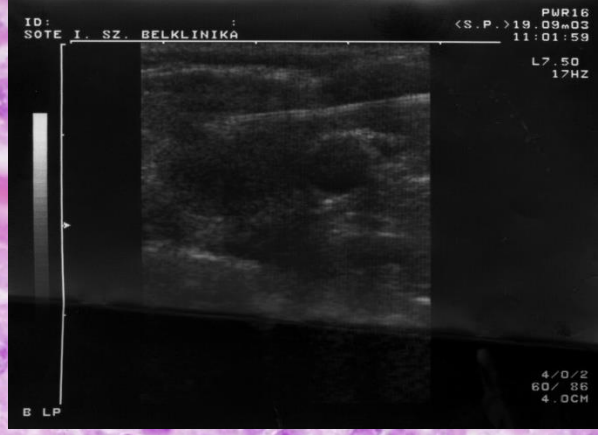


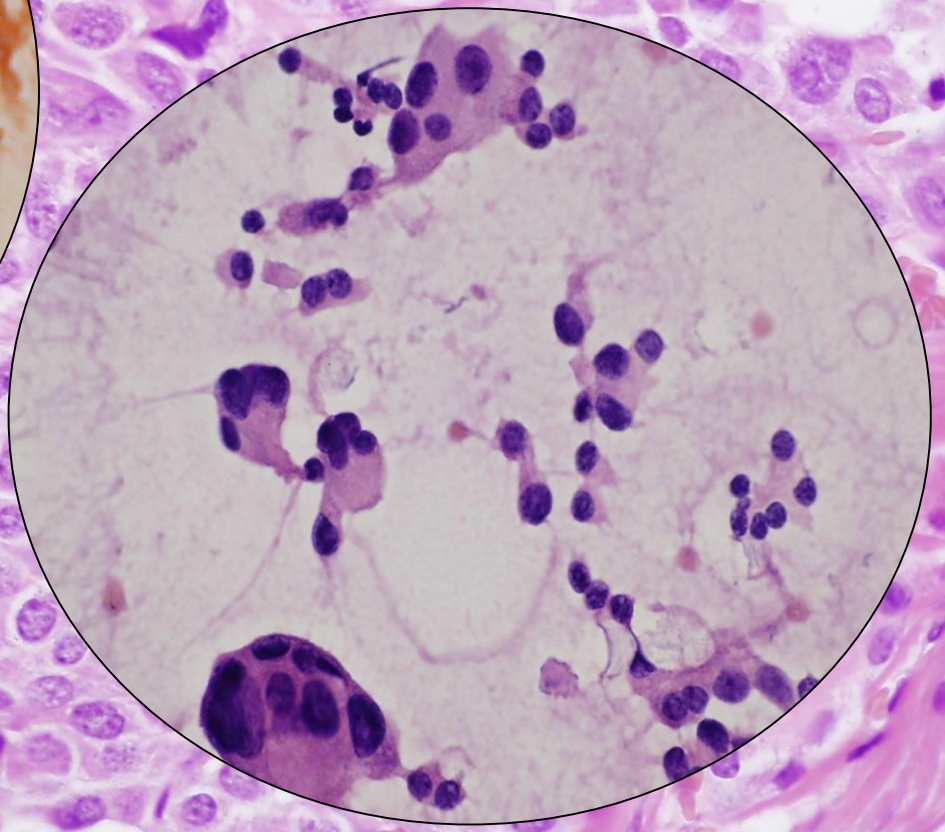




11109/07

55 y o
female





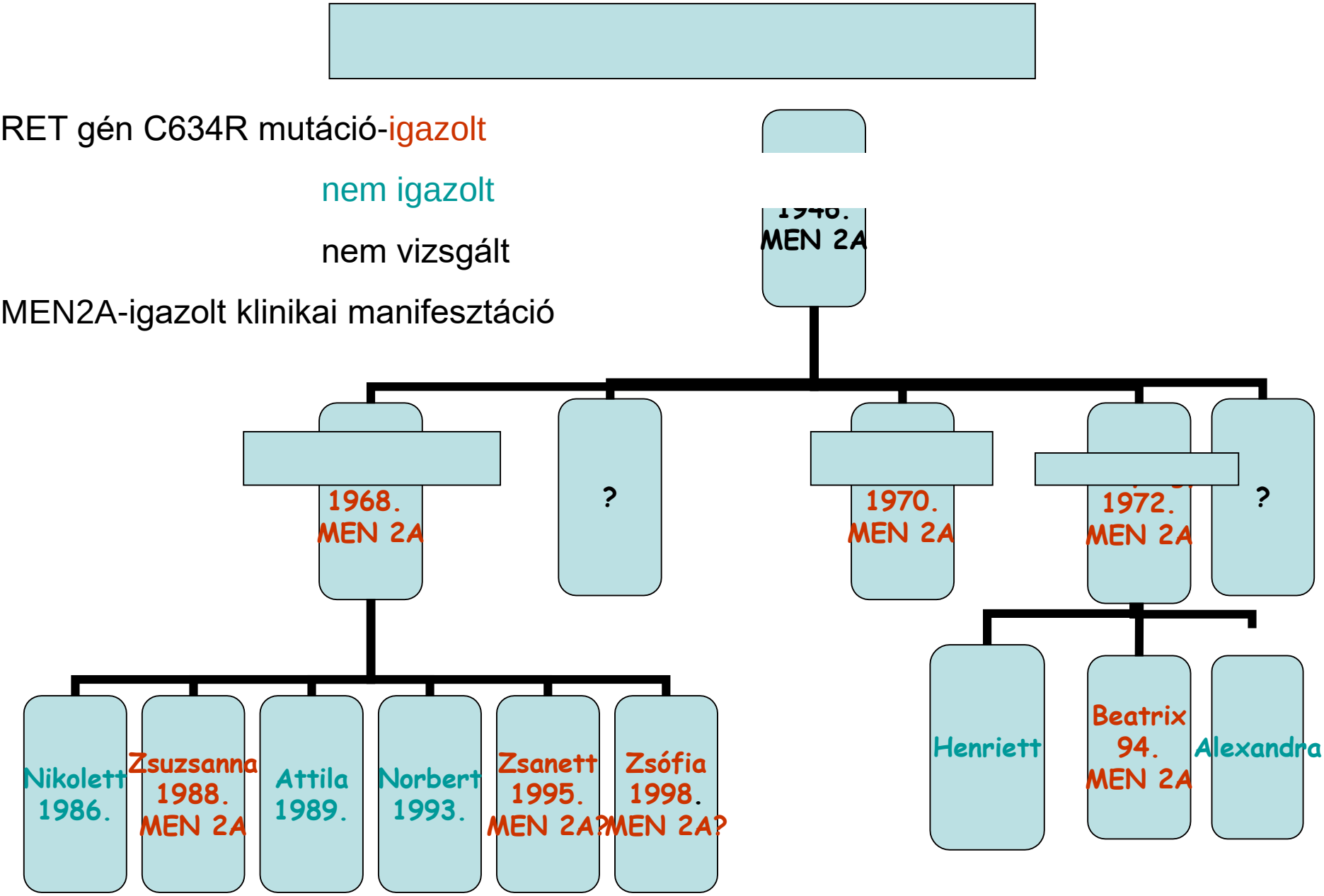
	MEN1 Wermer sy	MEN 2A Sipple sy	MEN 2B
Hypophysis	Adenomas		
parathyroid	HPL +++ Adenoma +	Hpl +	
Langerhans islands	HPL ++ Adenoma ++ CC +++		
Adrenal gland	HPL	Pheochromo-cytoma ++	Pheochromo-cytoma +++
Thyroid gland		C cell hpl +++ Medull. Cc +++	C cell hpl +++ Medull. Cc +++
Extraendocrine organs			Mucocutan ganglioneuromas Marfanoid stature
Genetic alteration	MEN1 11q13	RET 10q11.1	RET ?

RET gén C634R mutáció-igazolt

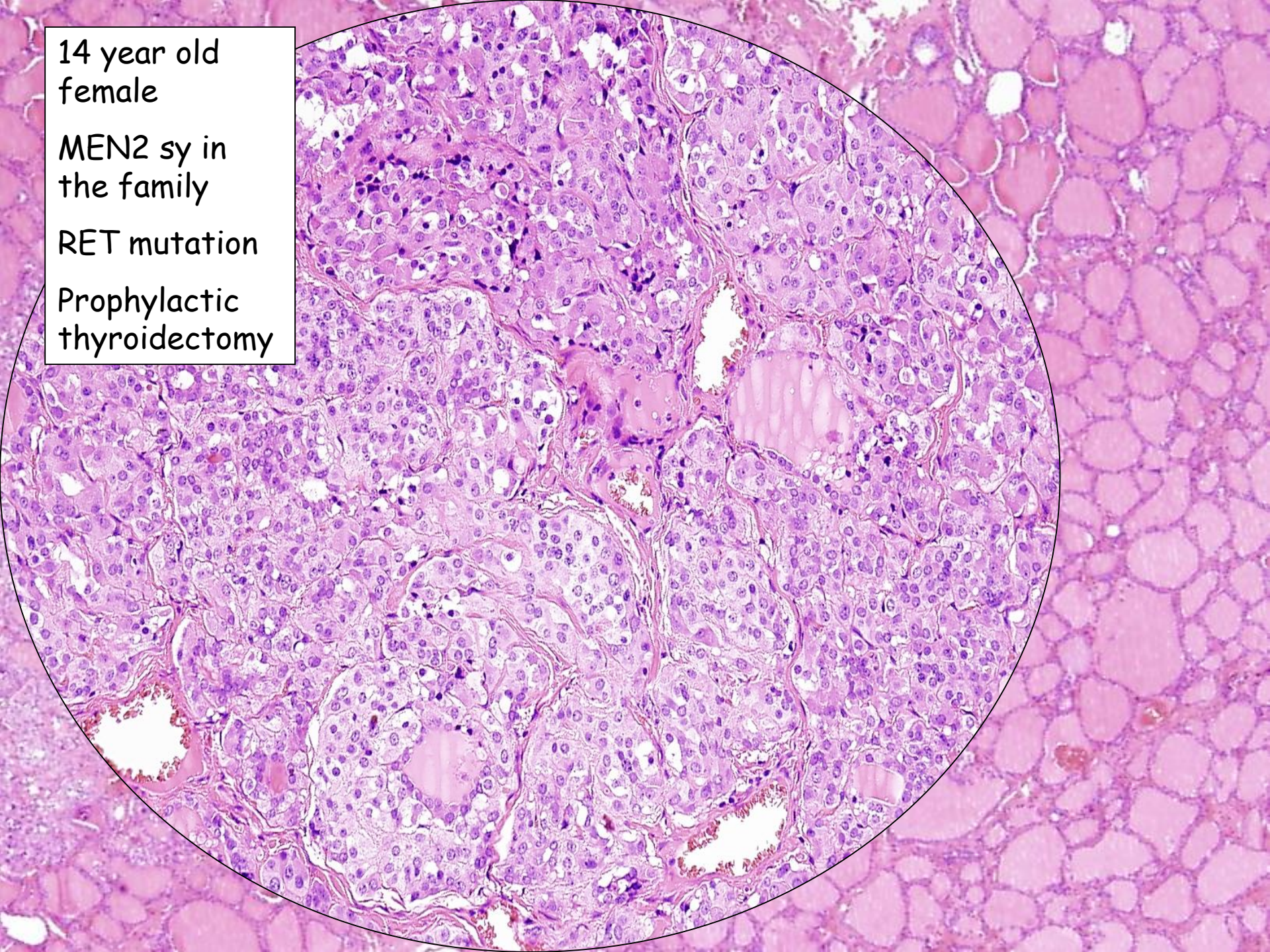
nem igazolt

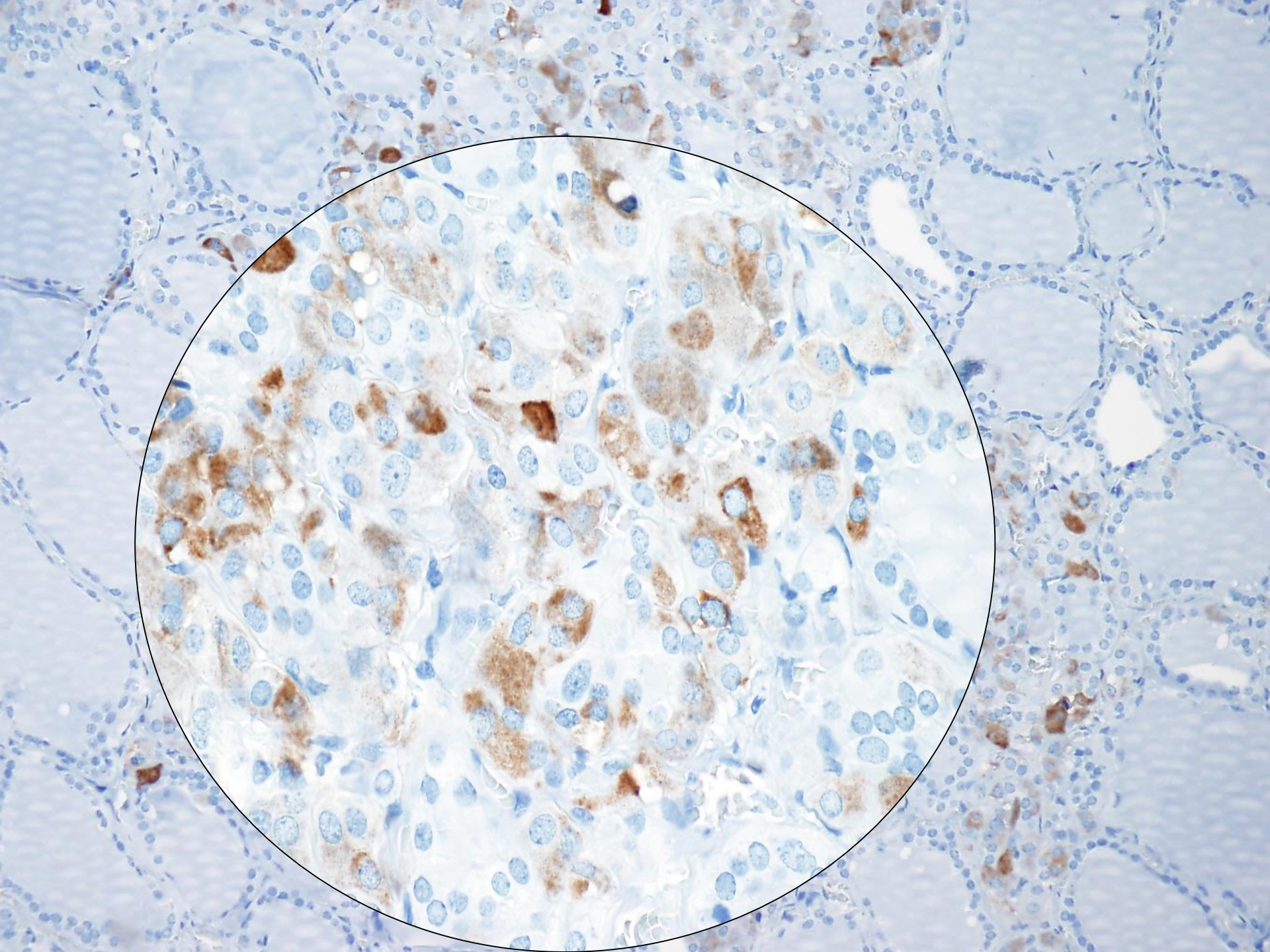
nem vizsgált

MEN2A-igazolt klinikai manifesztáció




2790/09 II/1


14 year old female
MEN2 sy in the family
RET mutation
Prophylactic thyroidectomy

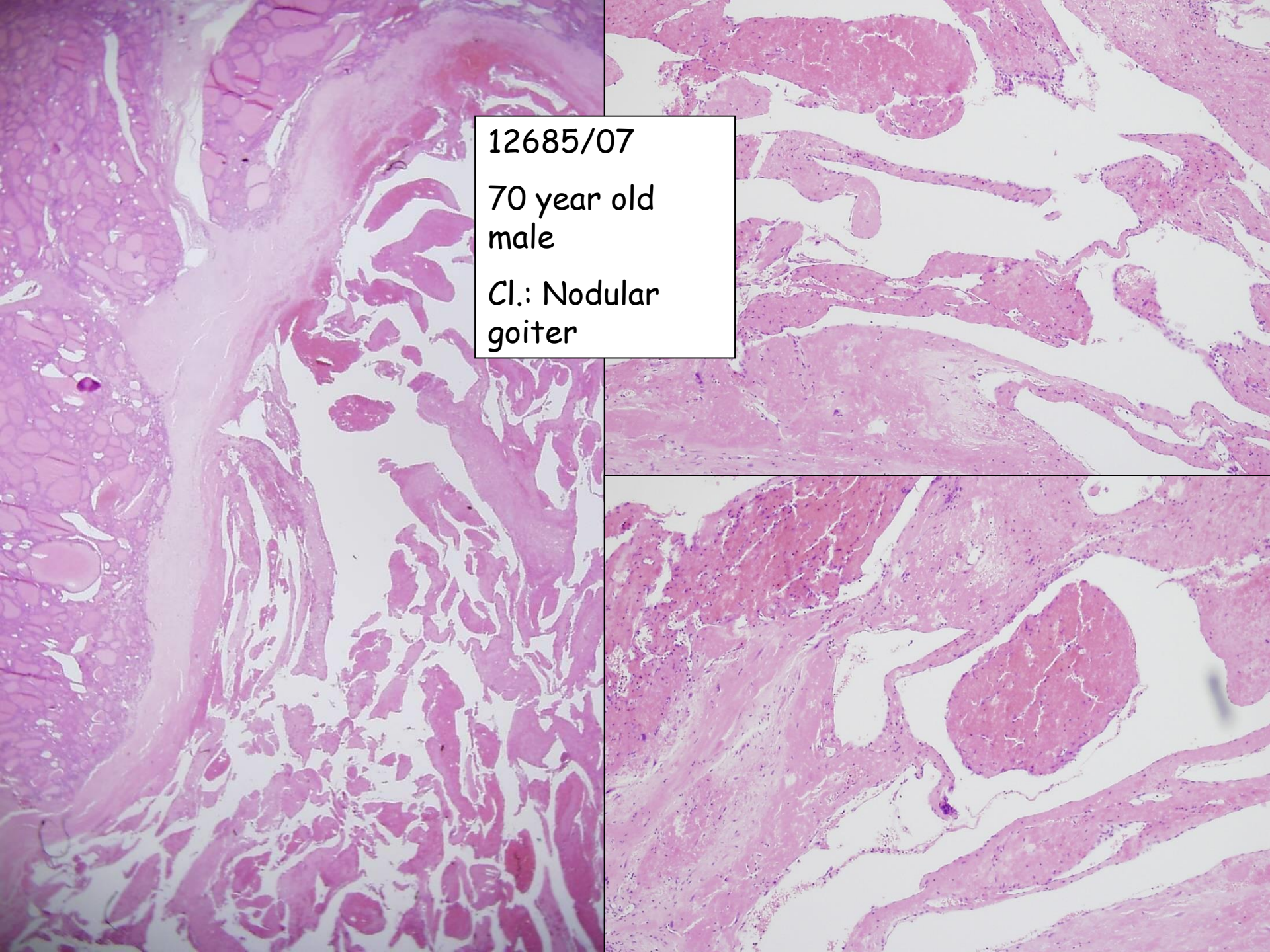


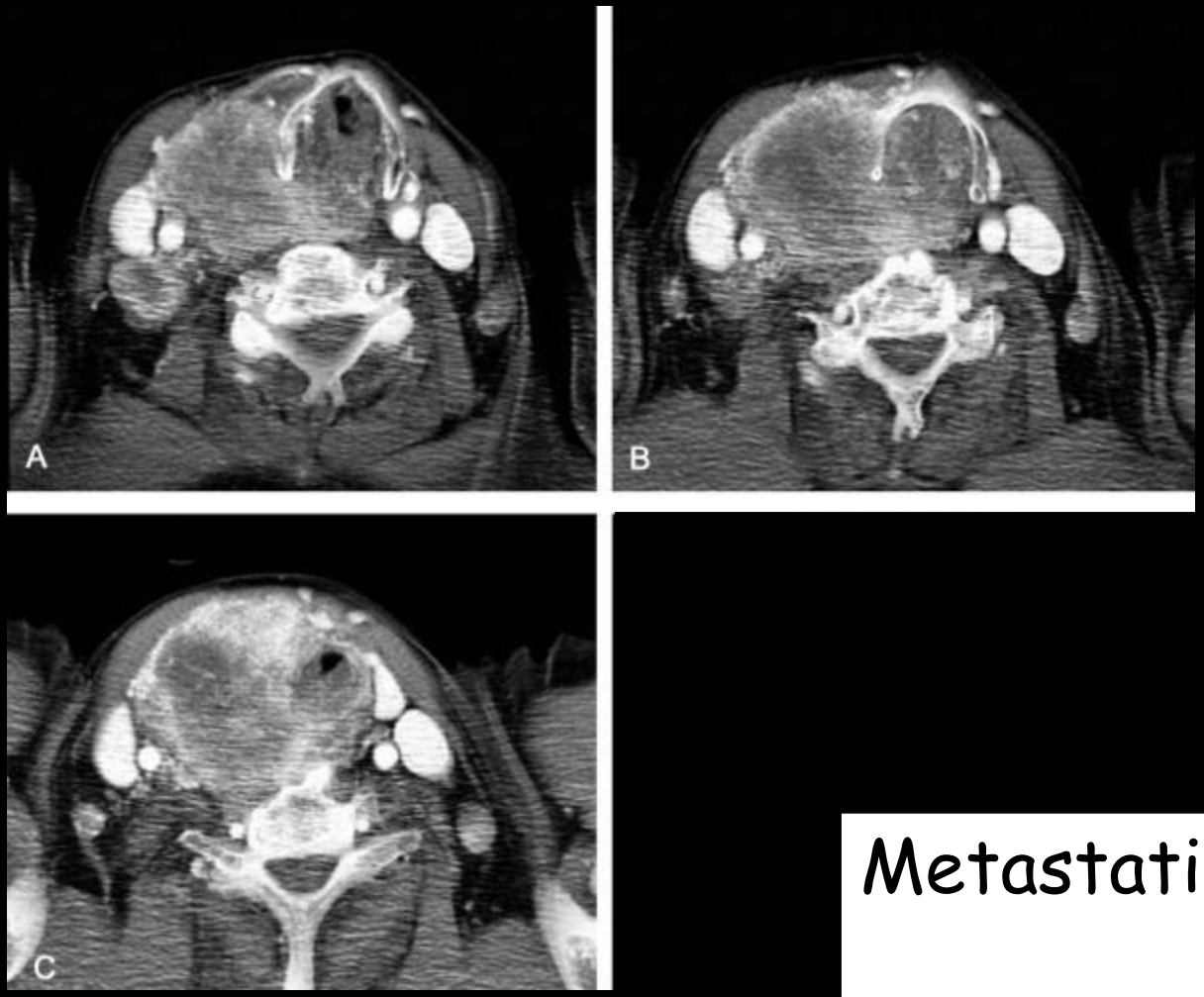
Other tumors

Mesenchymal tumors

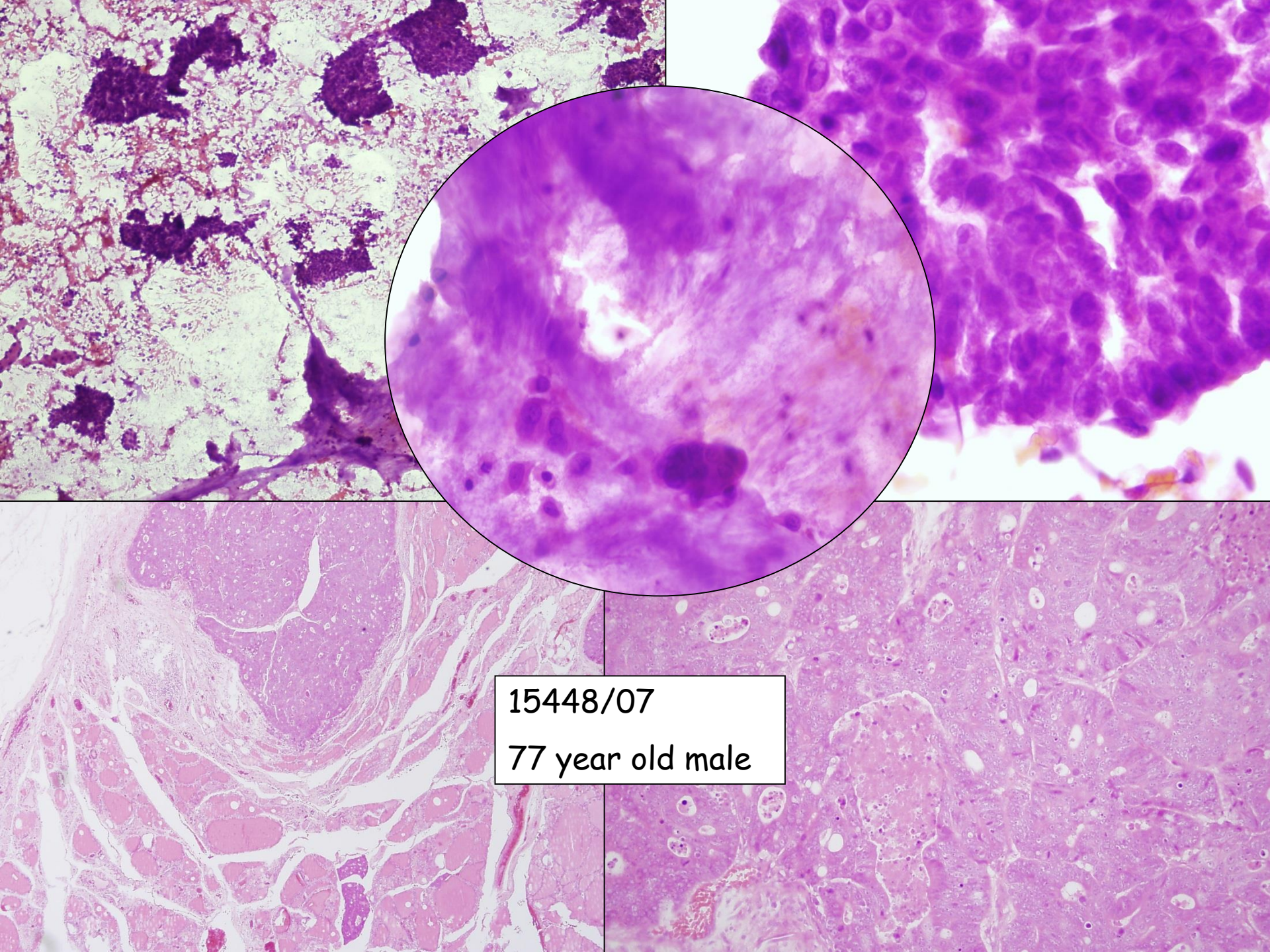
Lymphomas

Metastatic (rare)


12685/07
70 year old male
Cl.: Nodular goiter



Metastatic

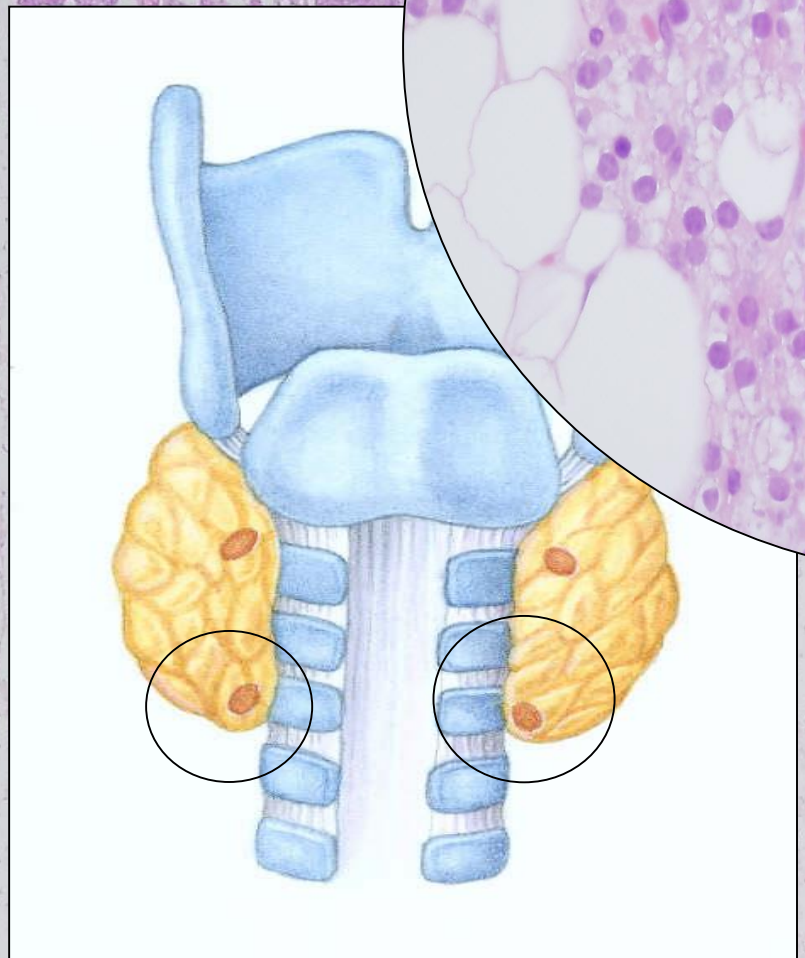

15448/07
77 year old male

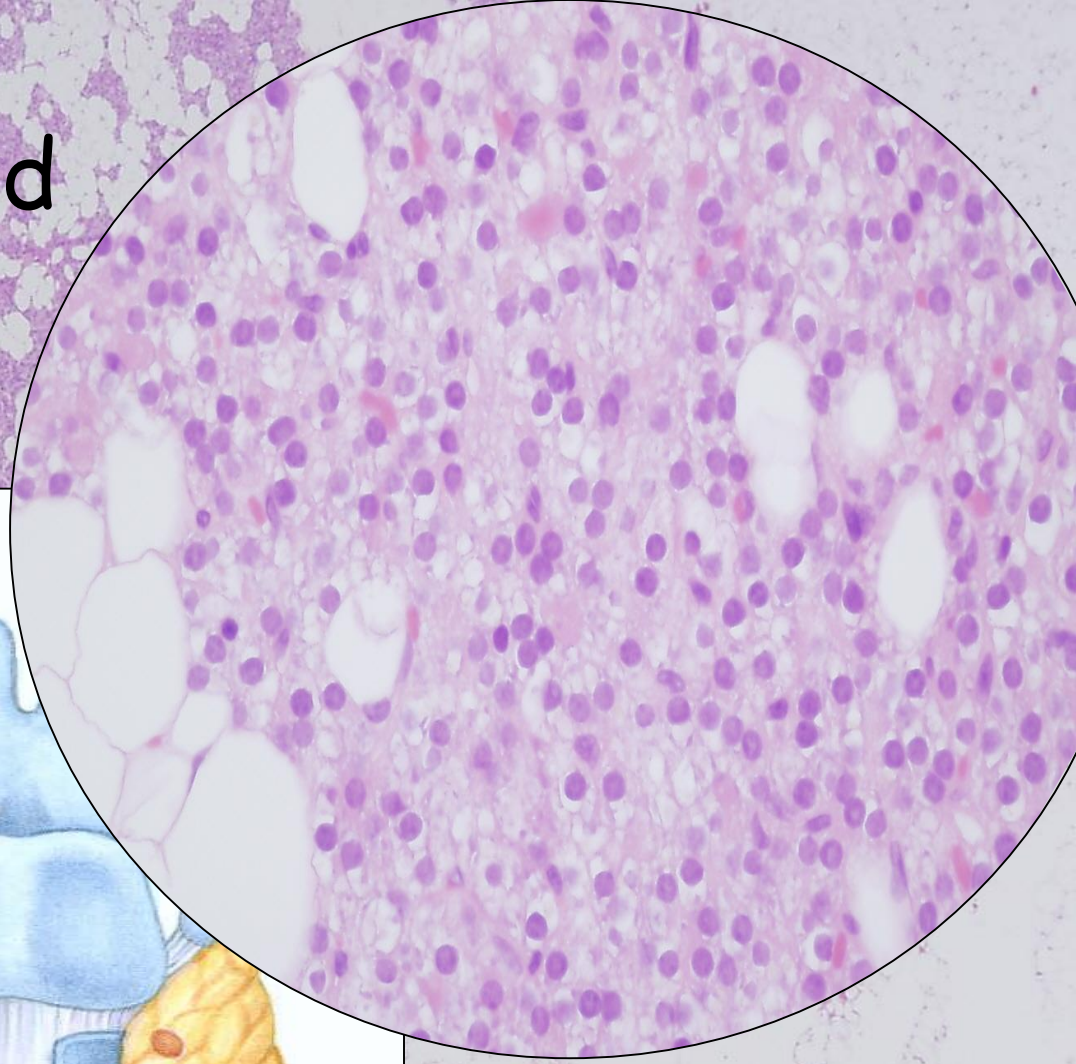
Parathyroid gland

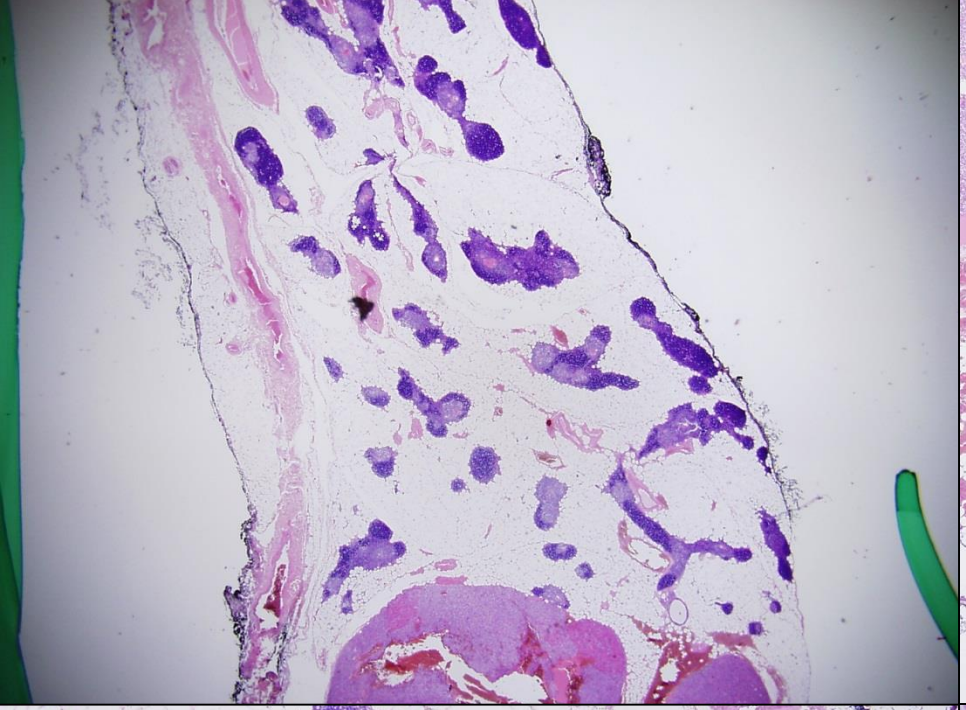
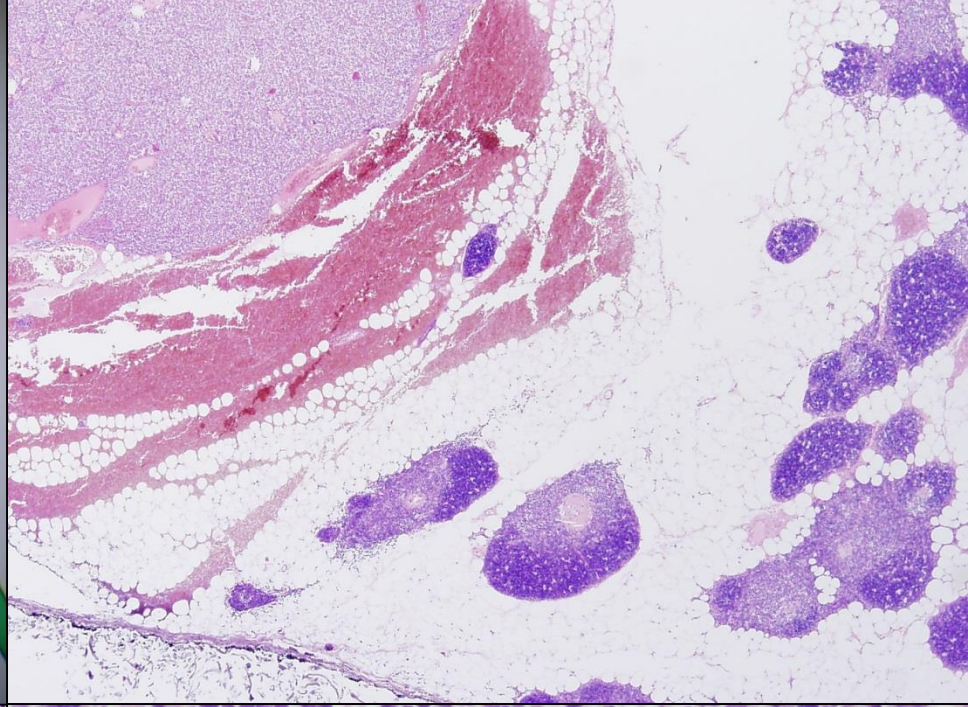
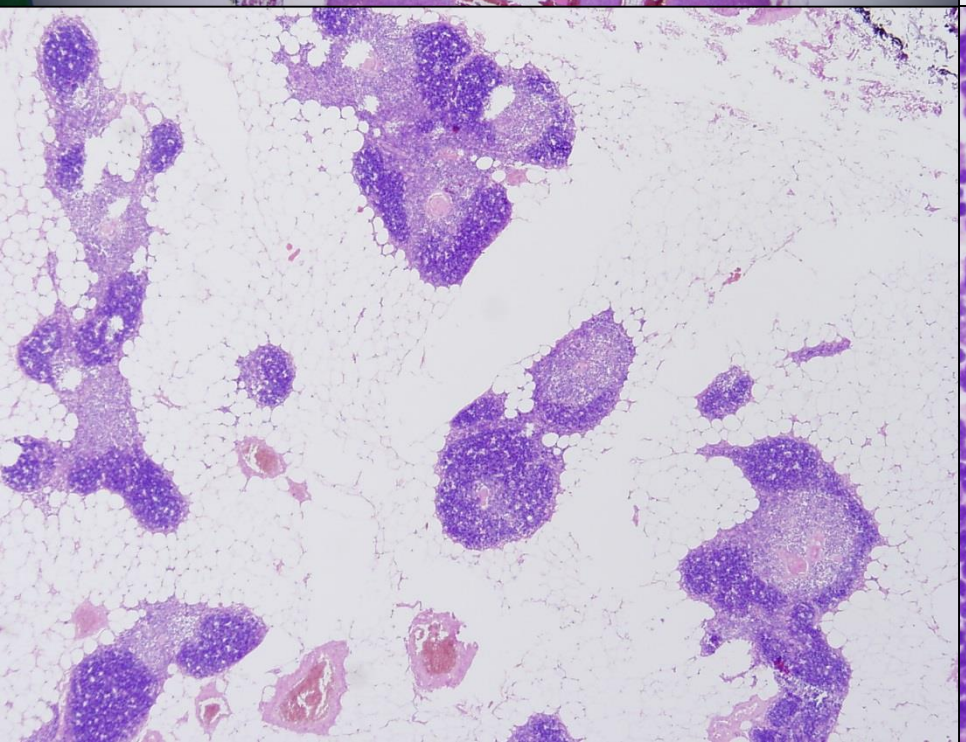
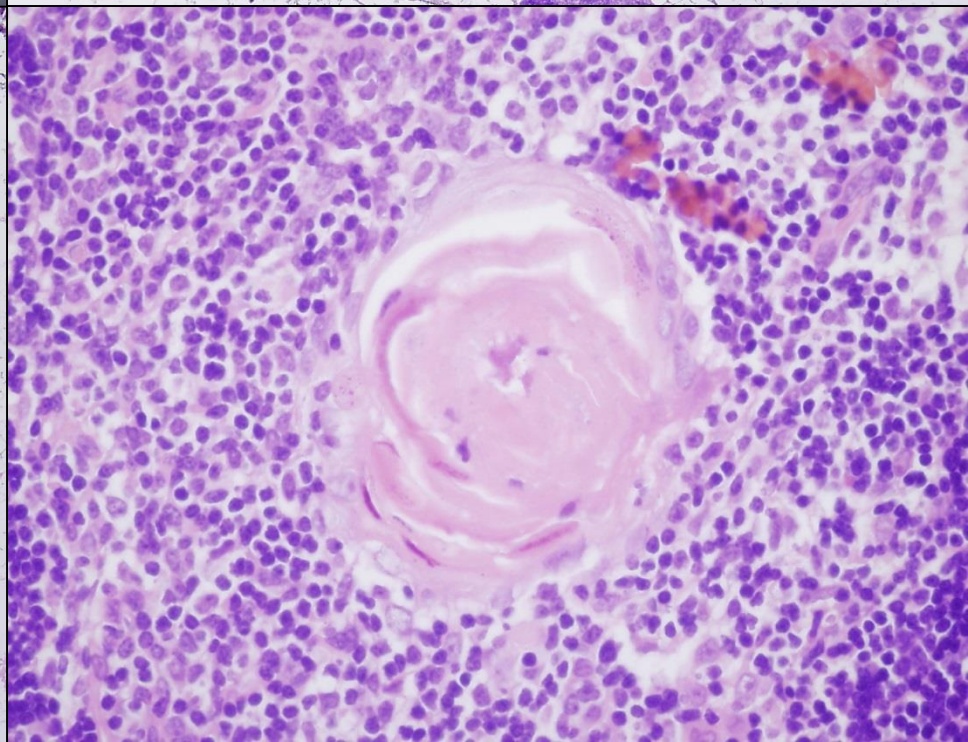
IV. Pharyngeal pouch

In 10 % of the cases, only 3 glands

Any localisation along the developmental pathway:





Norm.: 10-60 pg/ml or 1-6 pmol/l

Effects of the Parathyroid hormone …

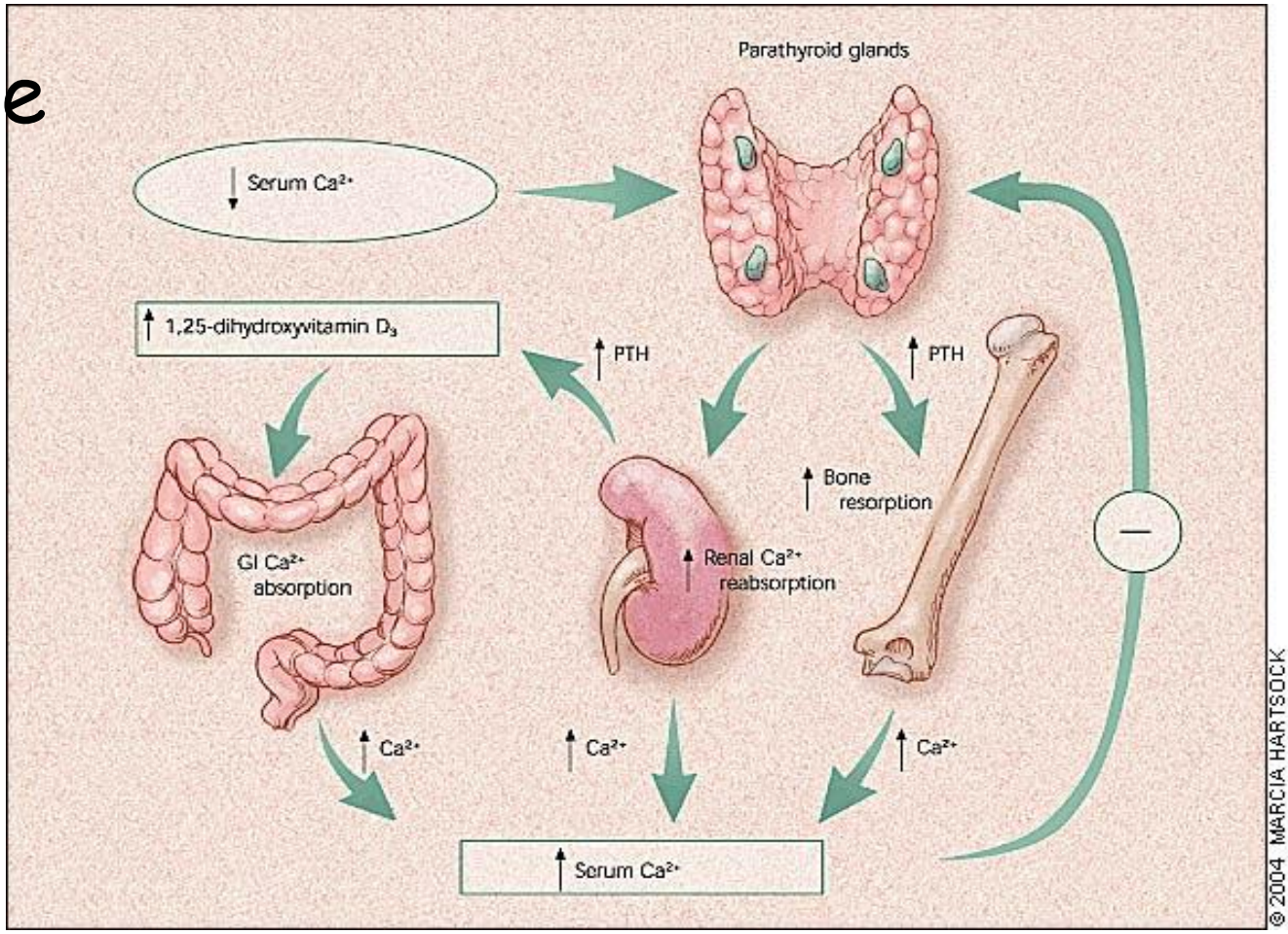
Osteoclast mobilization

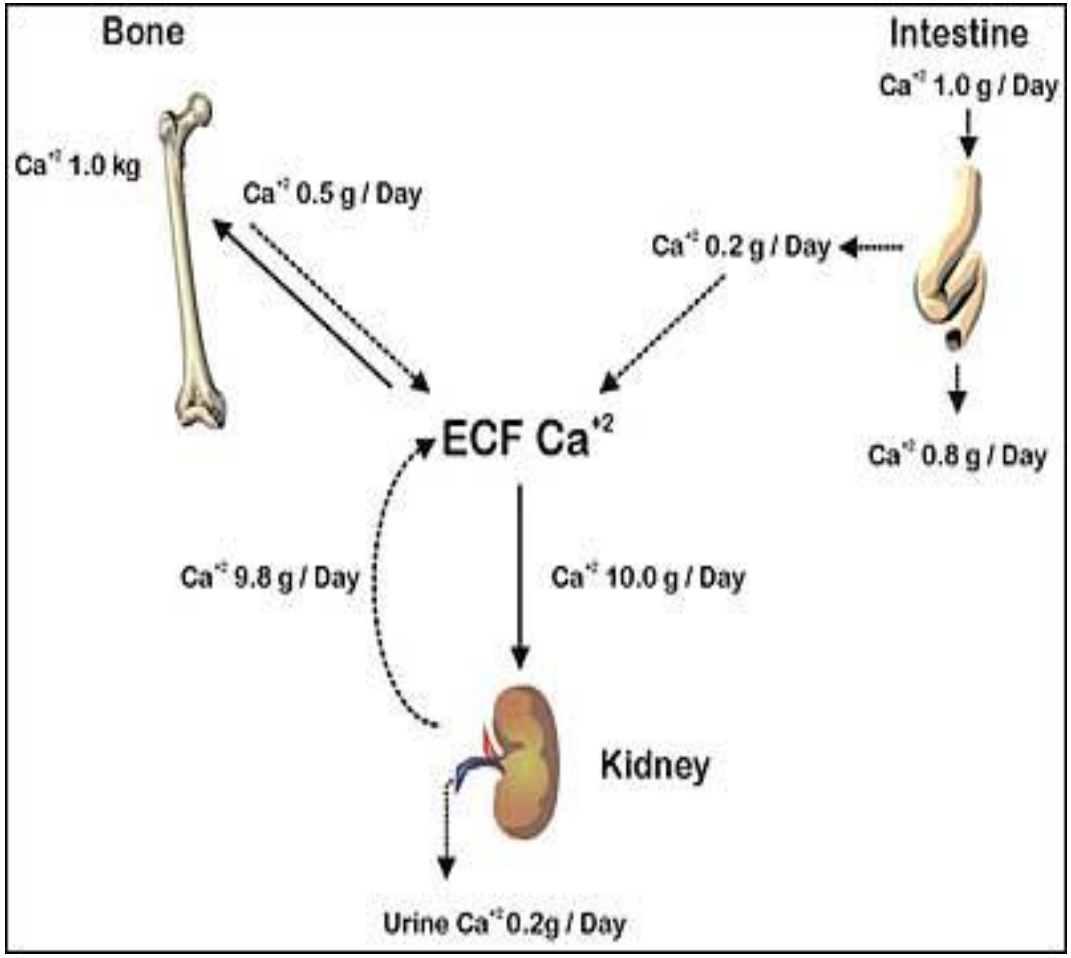
Renal tubular Ca reabsorbtion

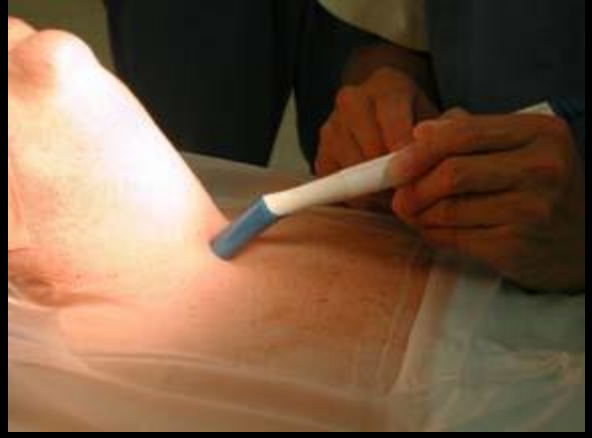
Renal vitamine D conversion (dihydroxy)
Renal phosphate

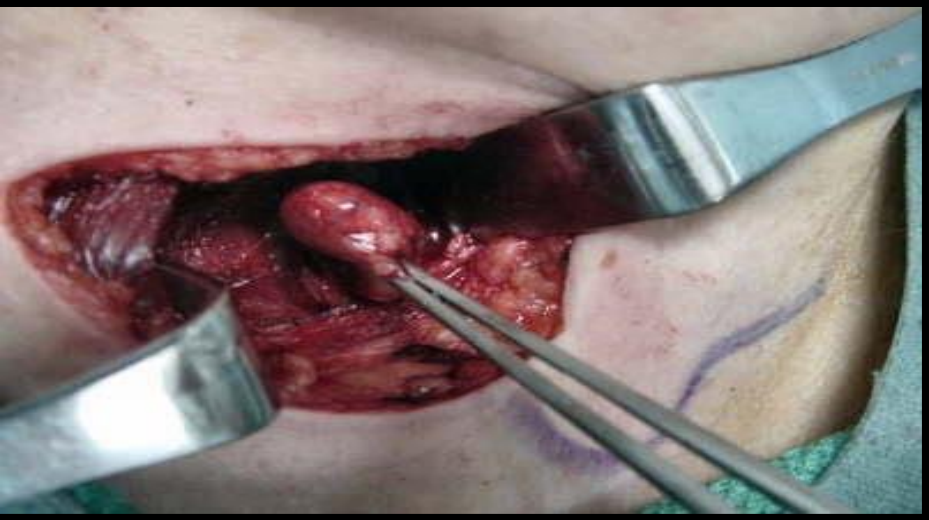
excretion ^

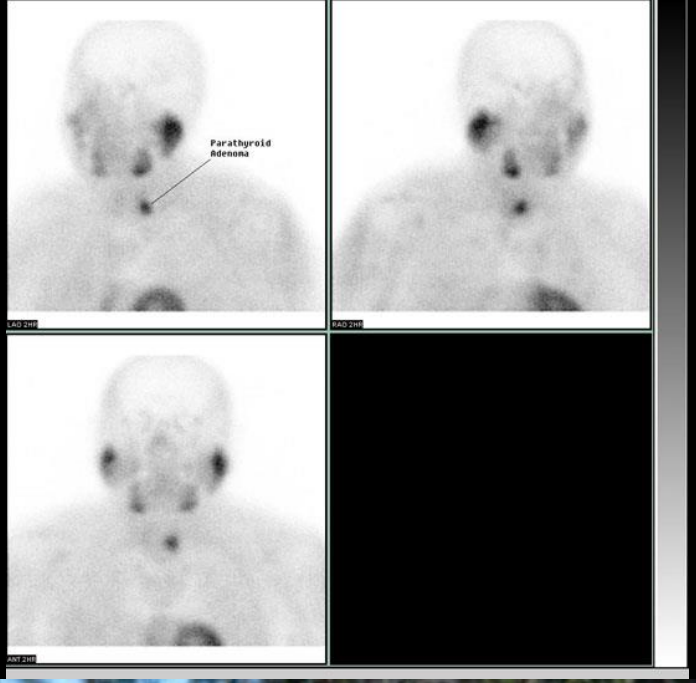
GI.: Ca absorption^




Bone
Intestine
Ca+2 1.0 g / Day
Ca+2 1.0 kg
Ca+2 0.5 g / Day
Ca+2 0.2 g / Day
ECF Ca+2
Ca+2 0.8 g / Day
Ca+2 9.8 g / Day
Ca+2 10.0 g / Day
Kidney
Urine Ca+2 0.2g / Day

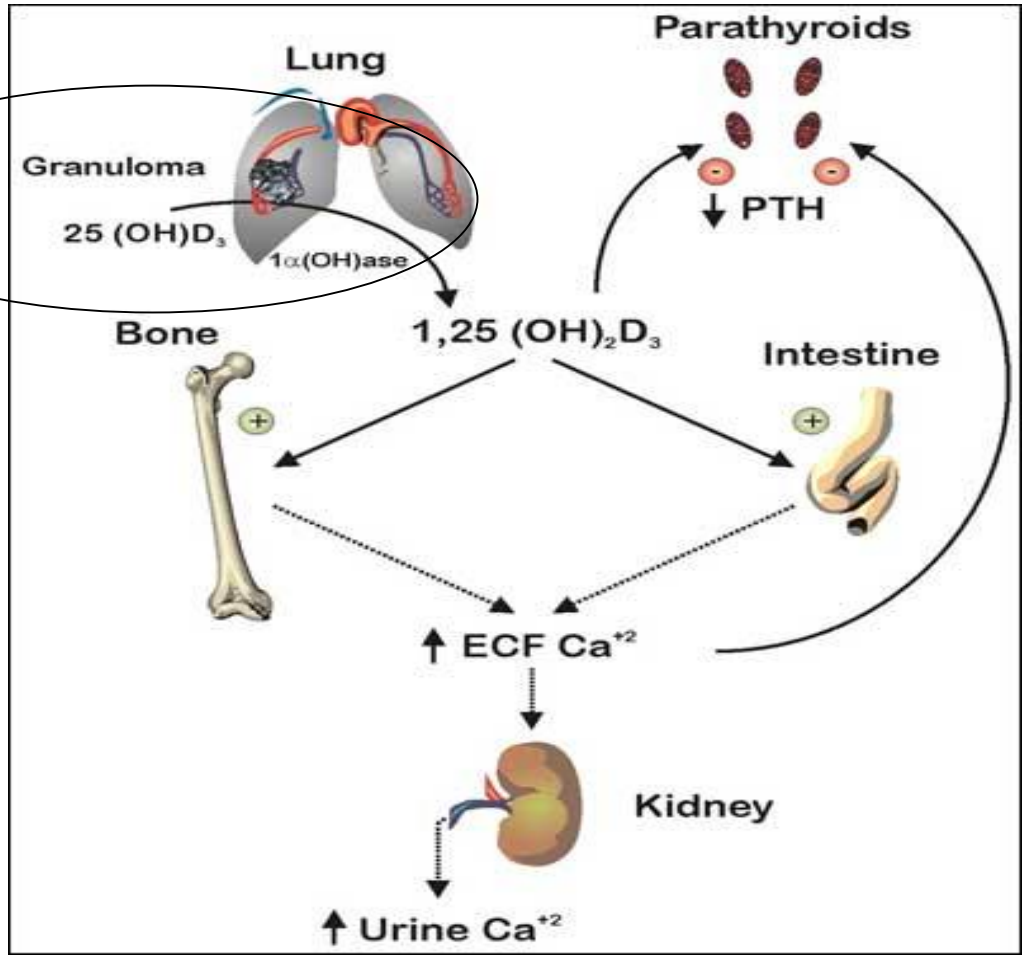






35-40 mg/piece, yellow-brown

Macrophages

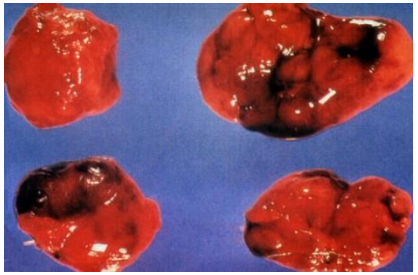


Primary hyperparathyreosis

parathyroid adenoma 75%

parathyroid hyperplasia 10→15%

parathyroid cc. <5%



Female/Male 3/1

Middle aged or older

Frequently sporadic, rarely part of MEN 1, or MEN2

MEN1 : 11q13 (tumor supressor gene inactivation)

MEN2A : 10 Q - RET mutation(tyrosine kinase receptor)

Familiary hypocalciuric syndrome:

3q (CASR) AD lowered sensitivity of the parathyroid for Ca

Sporadic- PRAD1:PRAD1 gene - coding CyclinD1- (11q) overexpression due to inversion , > clonal proliferation

	MEN1 Wermer sy	MEN 2A Sipple sy	MEN 2B
Hypophysis	Adenomas		
parathyroid	HPL +++ Adenoma +	Hpl +	
Langerhans ilands	HPL ++ Adenoma ++ CC +++		
Adrenal gland	HPL	Pheochromo-cytoma ++	Pheochromo-cytoma +++
Thyroid gland		C cell hpl +++ Medull. Cc +++	C cell hpl +++ Medull. Cc +++
Extraendocrine organs			Mucocutan ganglioneuromas Marfanoid stature
Genetic alteration	MEN1 11q13	RET 10q11.1	RET ?

Hyperparathyreosis

Asymptomatic

Blood test performed for unrelated conditions: Se Ca ^^^

associated with malignancy

Symptomatic,

Neuromuscular changes – weekness, fatigue

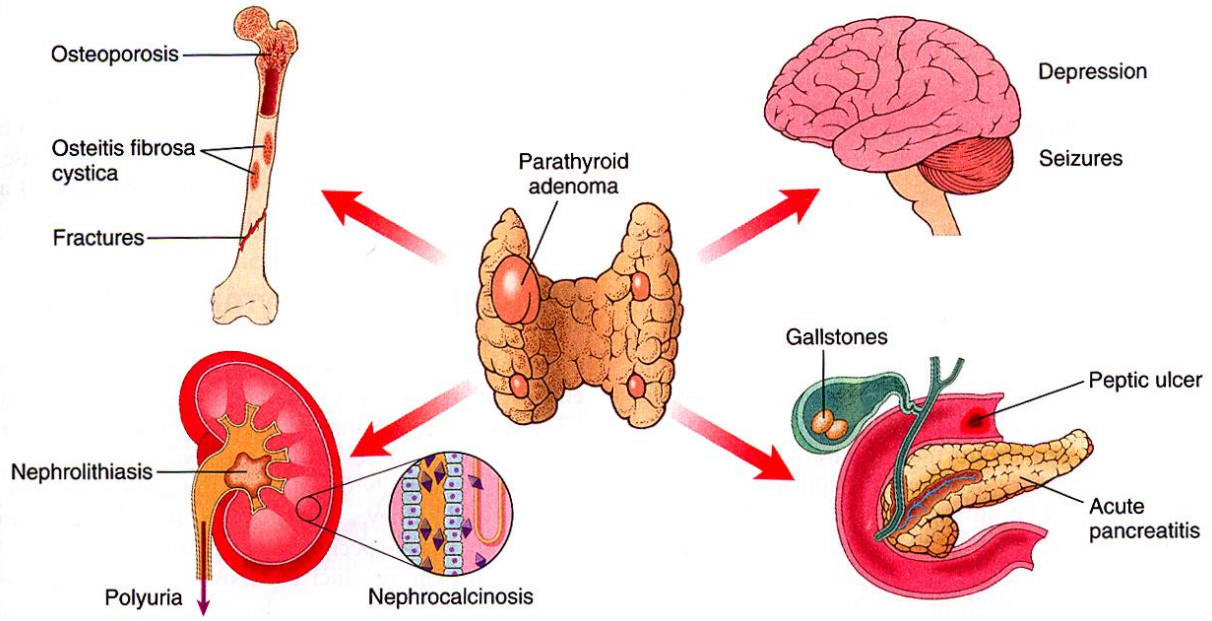
Cardial: aorta, mitral calcification

GI nausea, obstipation, ulcers, pancreatitis, gallstones

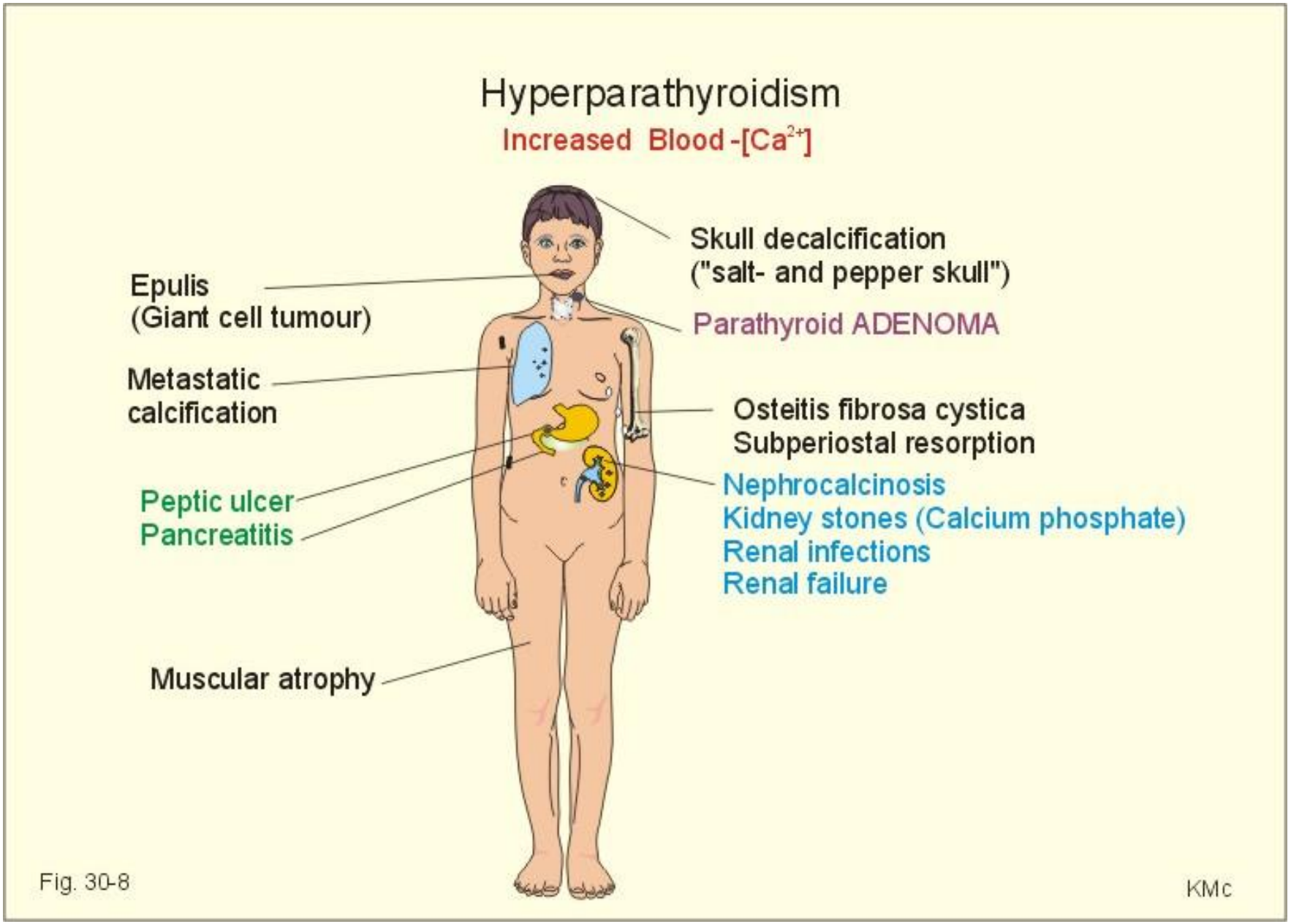
CNS depression, letargy, cramps

Bone diseases

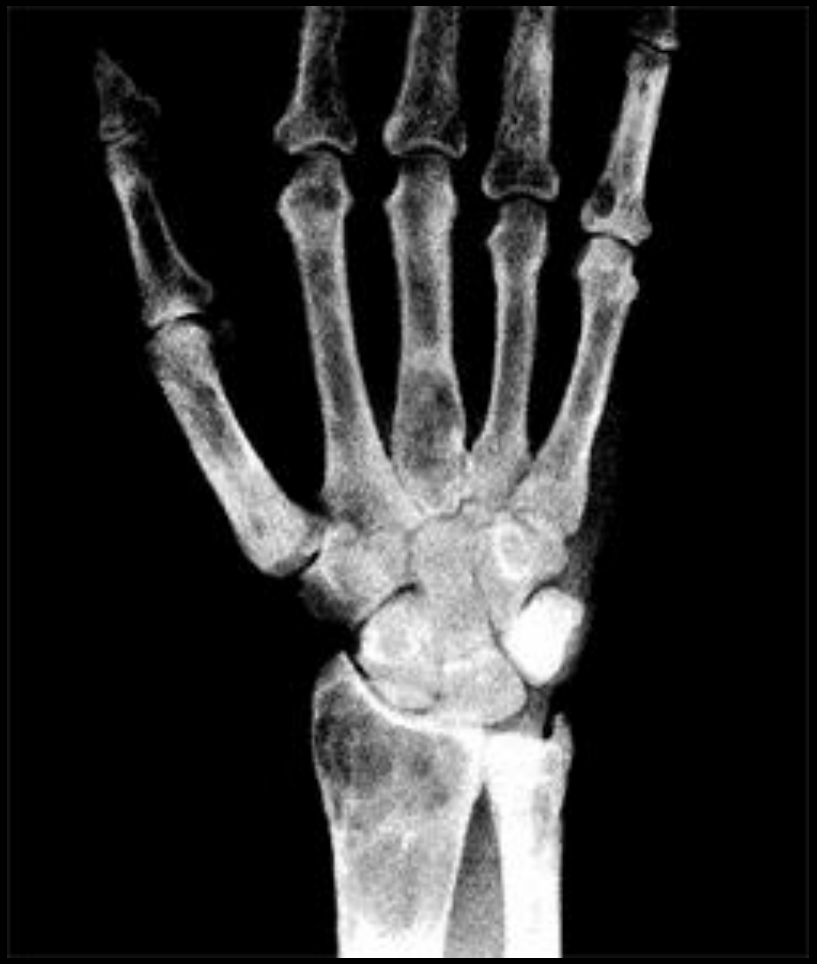
(osteitis fibrosa cystica generalista secundum Recklinghausen)

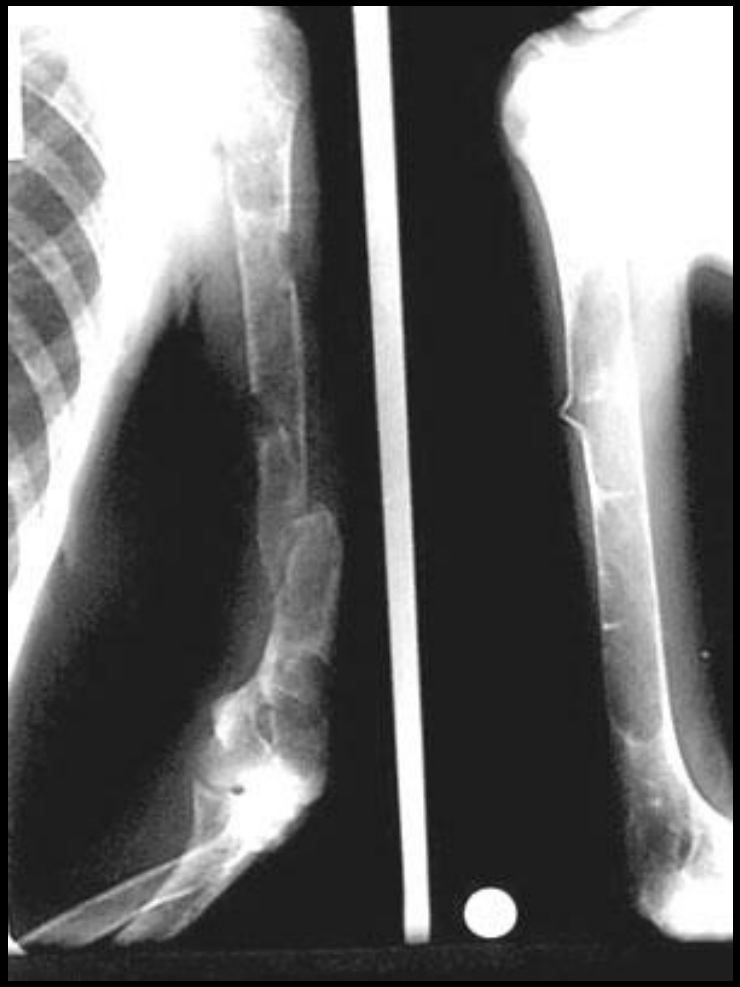


Sequales of Hyperparathyreosis



Osteitis fibrosa cystica generalisata secundum Recklinghausen

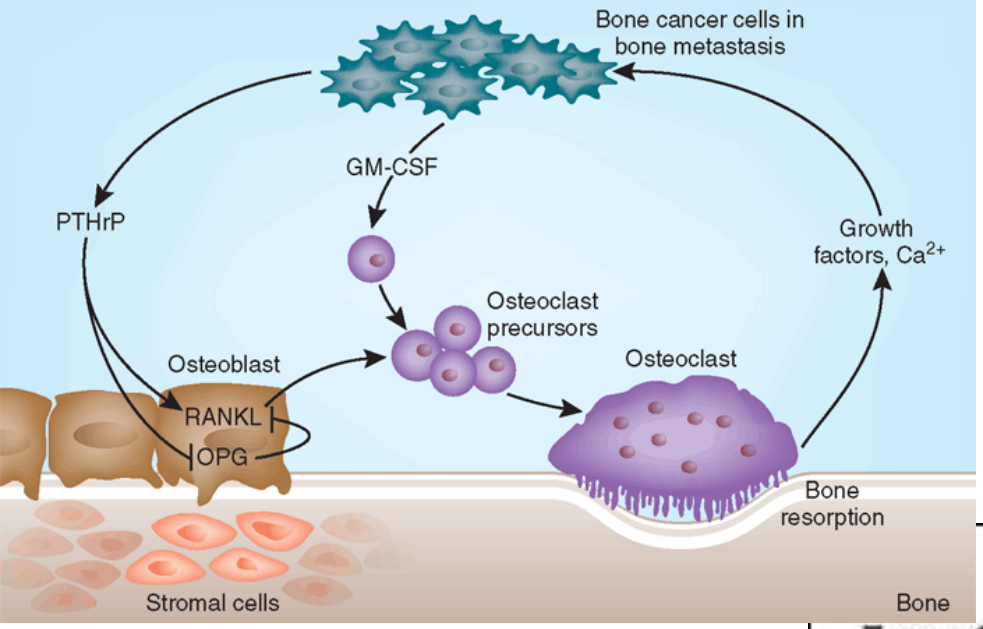




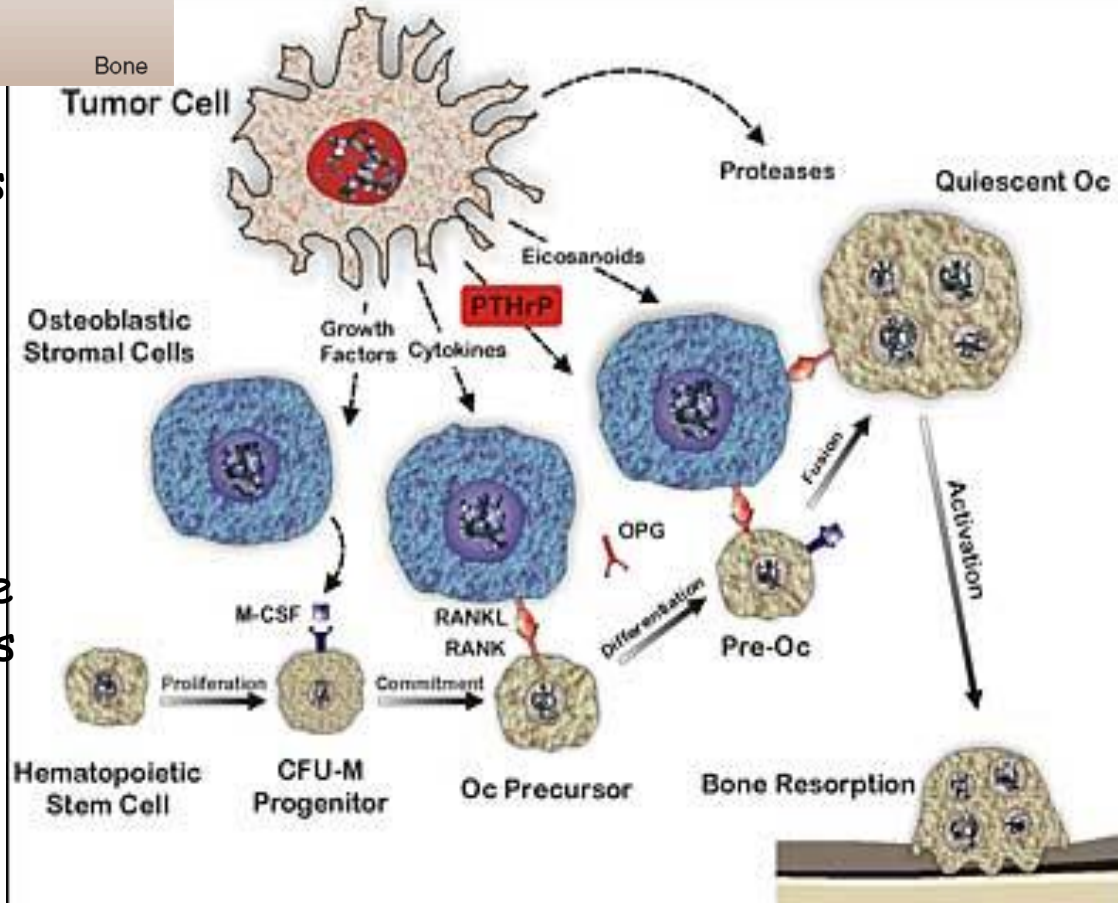
Causes of Hypercalcaemia

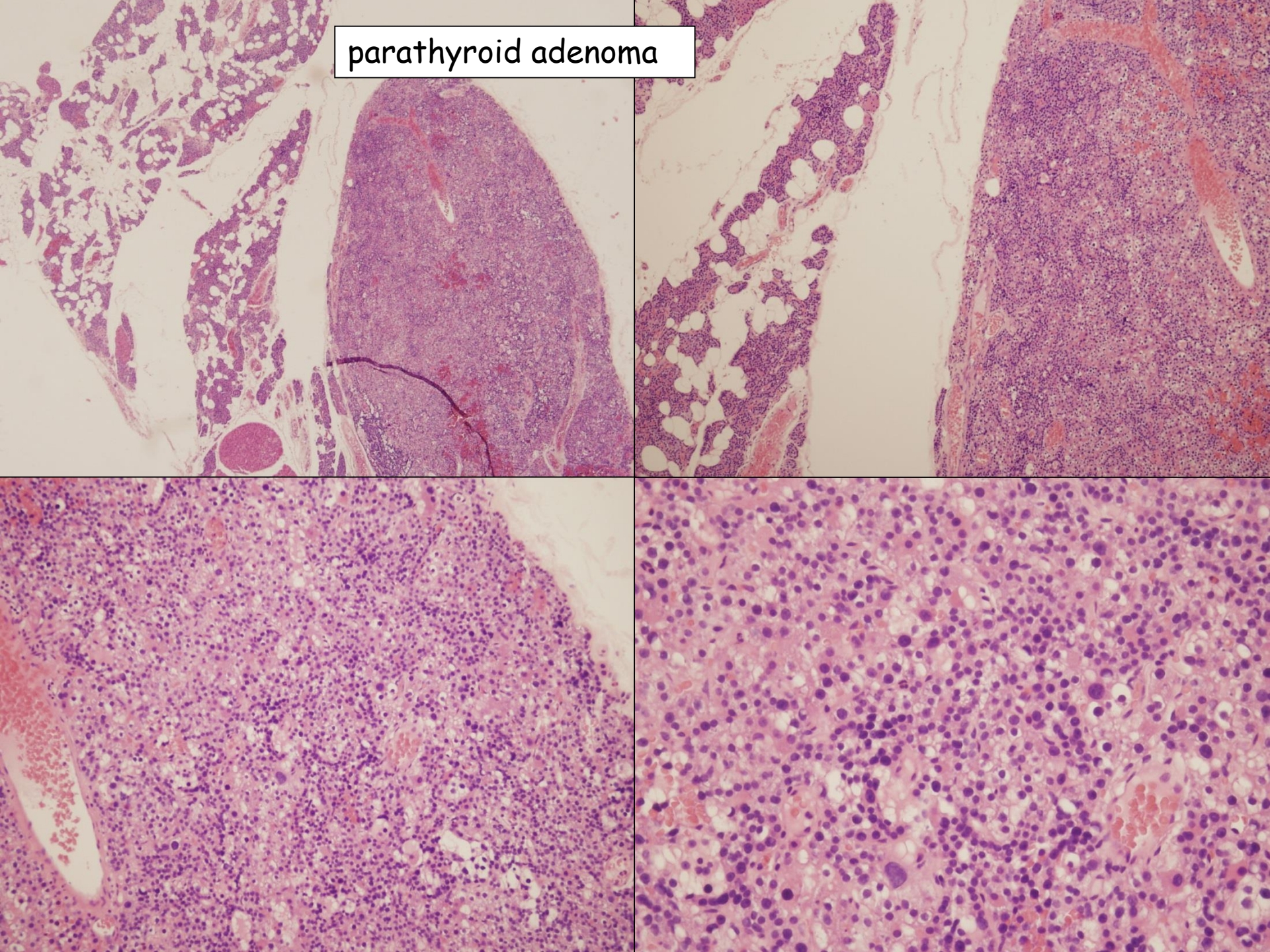
Elevated PTH
- Hyperparathyreosis
  - primary
  - secondary
  - tertiary

Lowered PTH
- Malignancy associated
  - Osteolytic met.
  - PTH-rP-mediated
- D vitamine toxicity
- Immobilization
- Thiazids
- Sarcoidosis (other granulomatous diseases)



GM-CSF expands the osteoclast precursor pool and PTHrP increases RANK ligand and decreases osteoprotegerin (OPG) production by osteoblasts; OPG is a decoy receptor that blocks RANKL. RANKL then induces osteoclast precursor differentiation and increases osteoclast formation. The increase in bone resorption releases growth factors and calcium, which then enhances tumor growth.




parathyroid adenoma

Microscopy

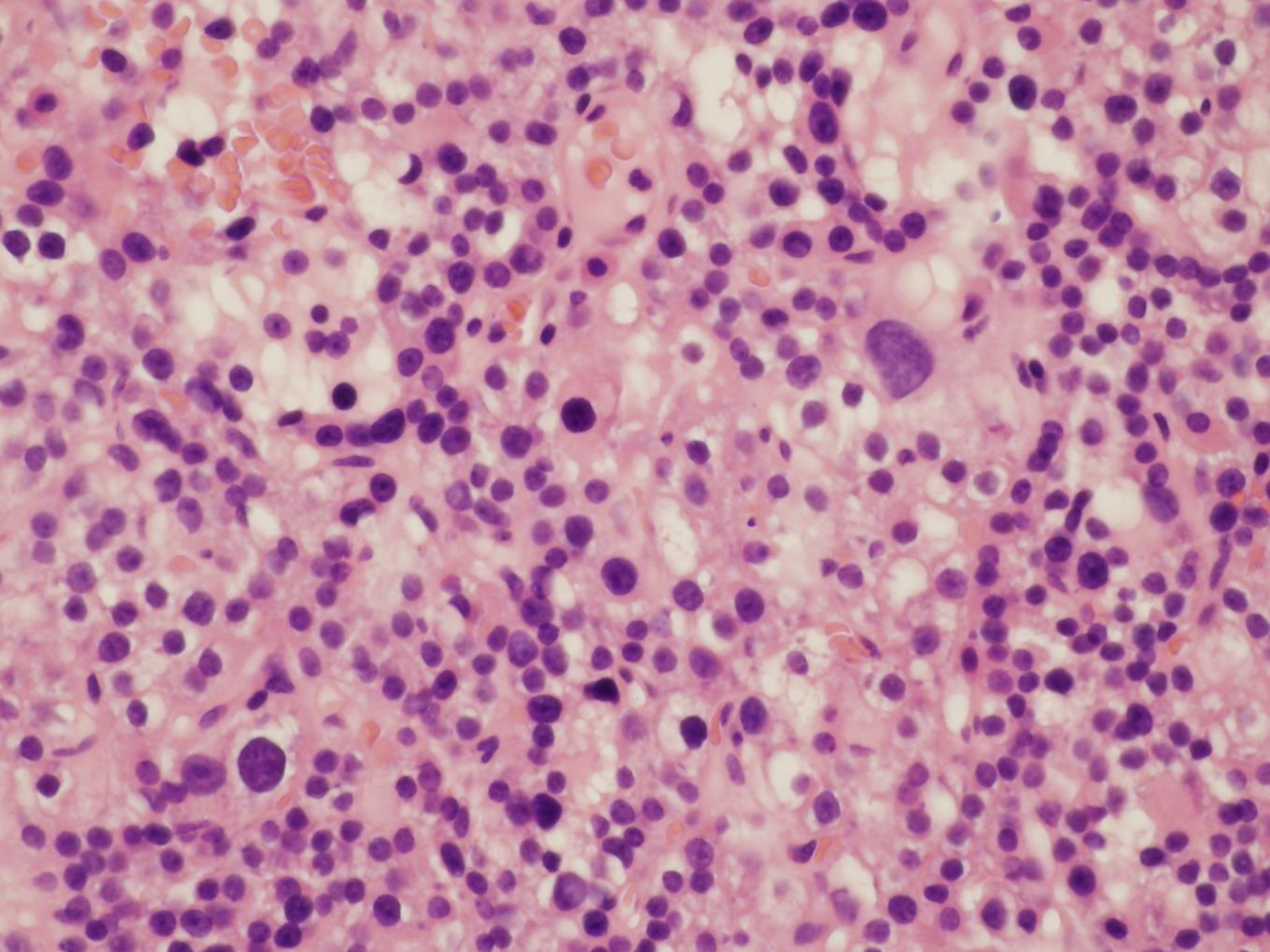
Normal looking parathyroid tissue surrounded by a capsule

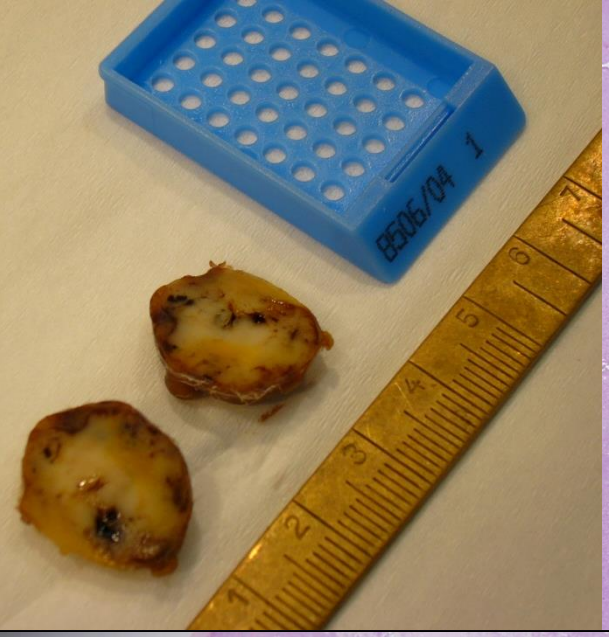
Endocrine atypia might occure

Adenoma:– 1 gland gets enlarged

Hyperplasia: – more glands get enlarged but not evidently all (??)

Carcinoma: diagnosis is based on vascular / capsular invasion, metastasis

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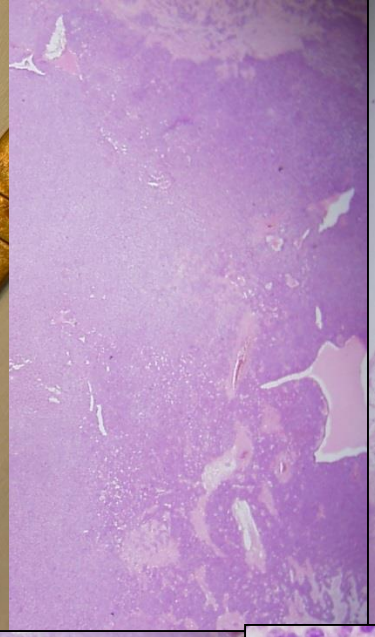
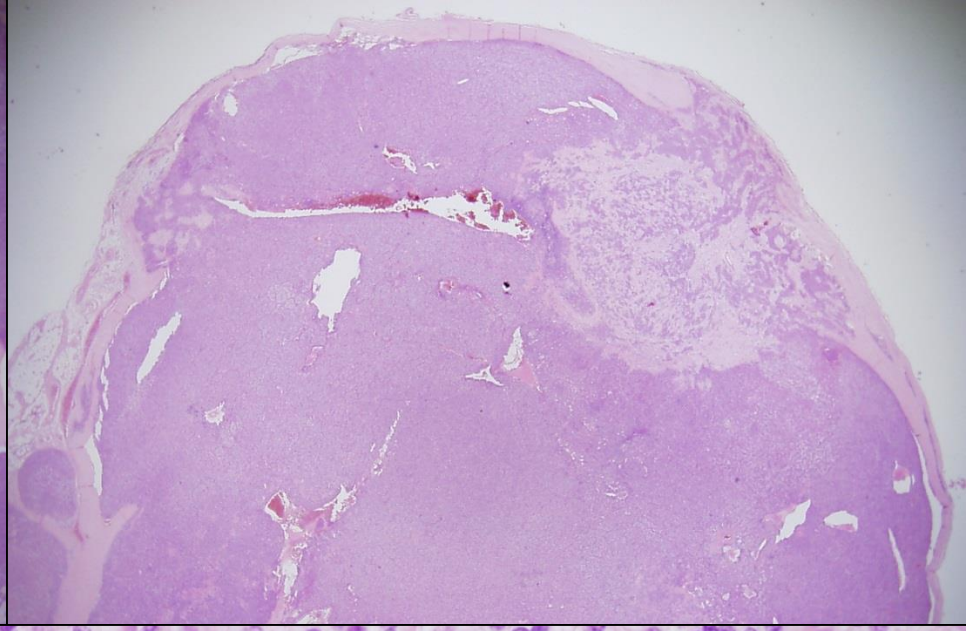
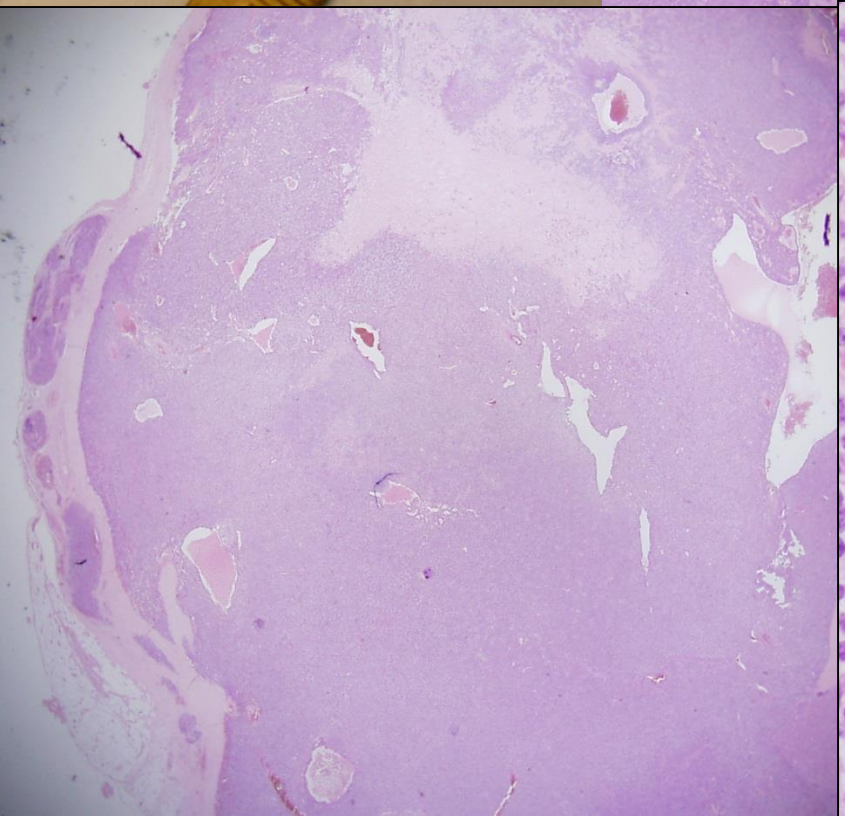
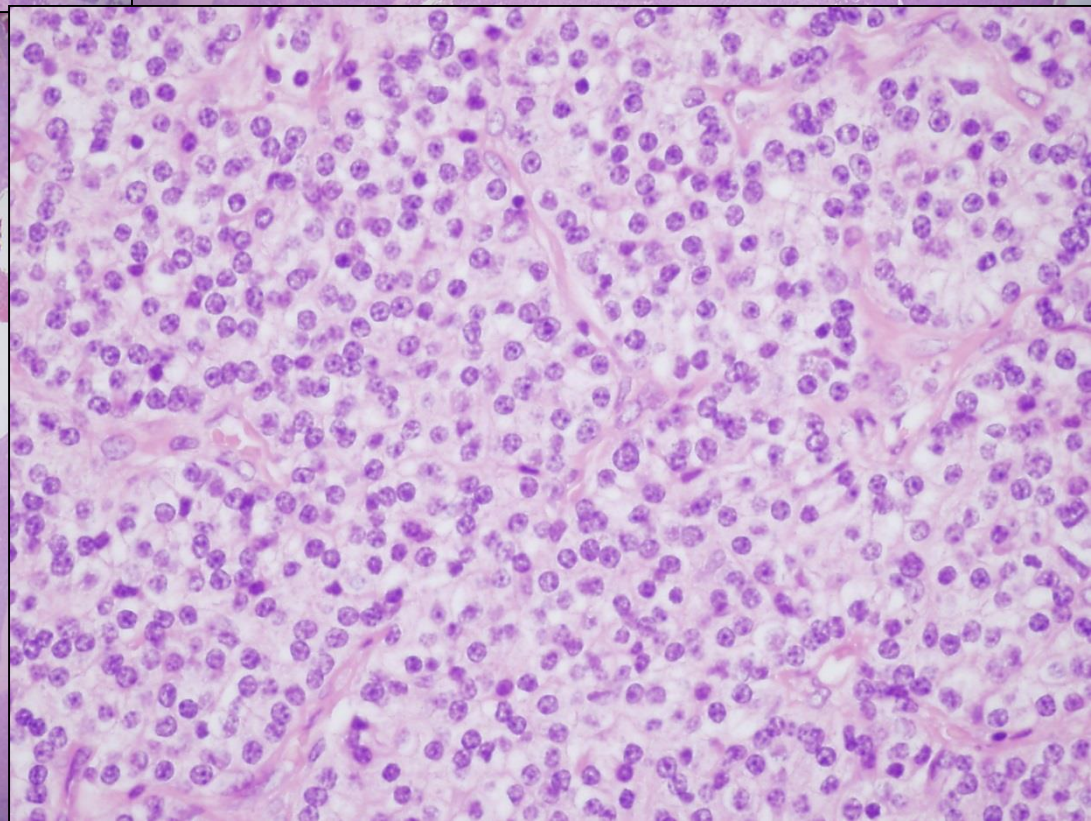
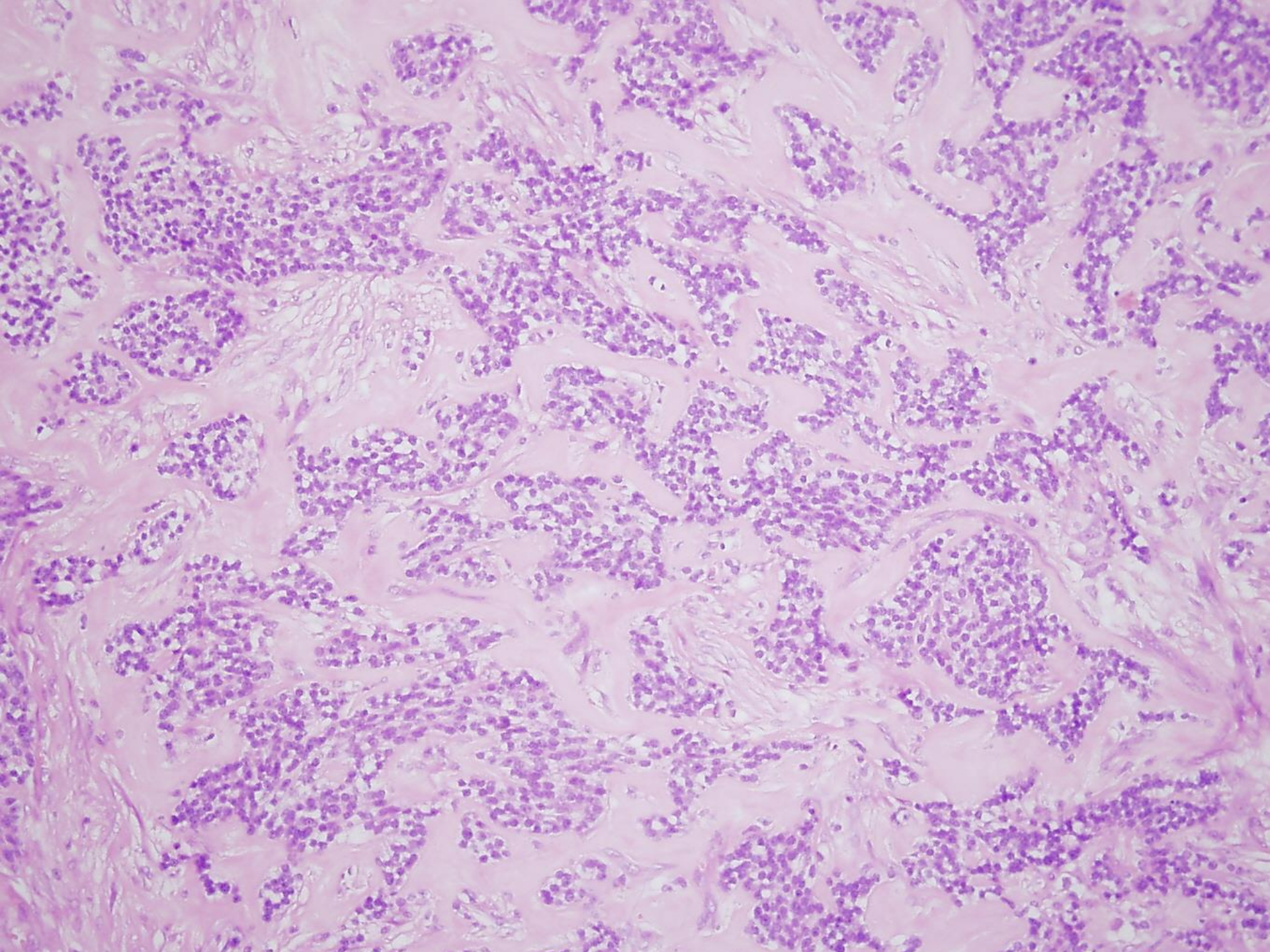
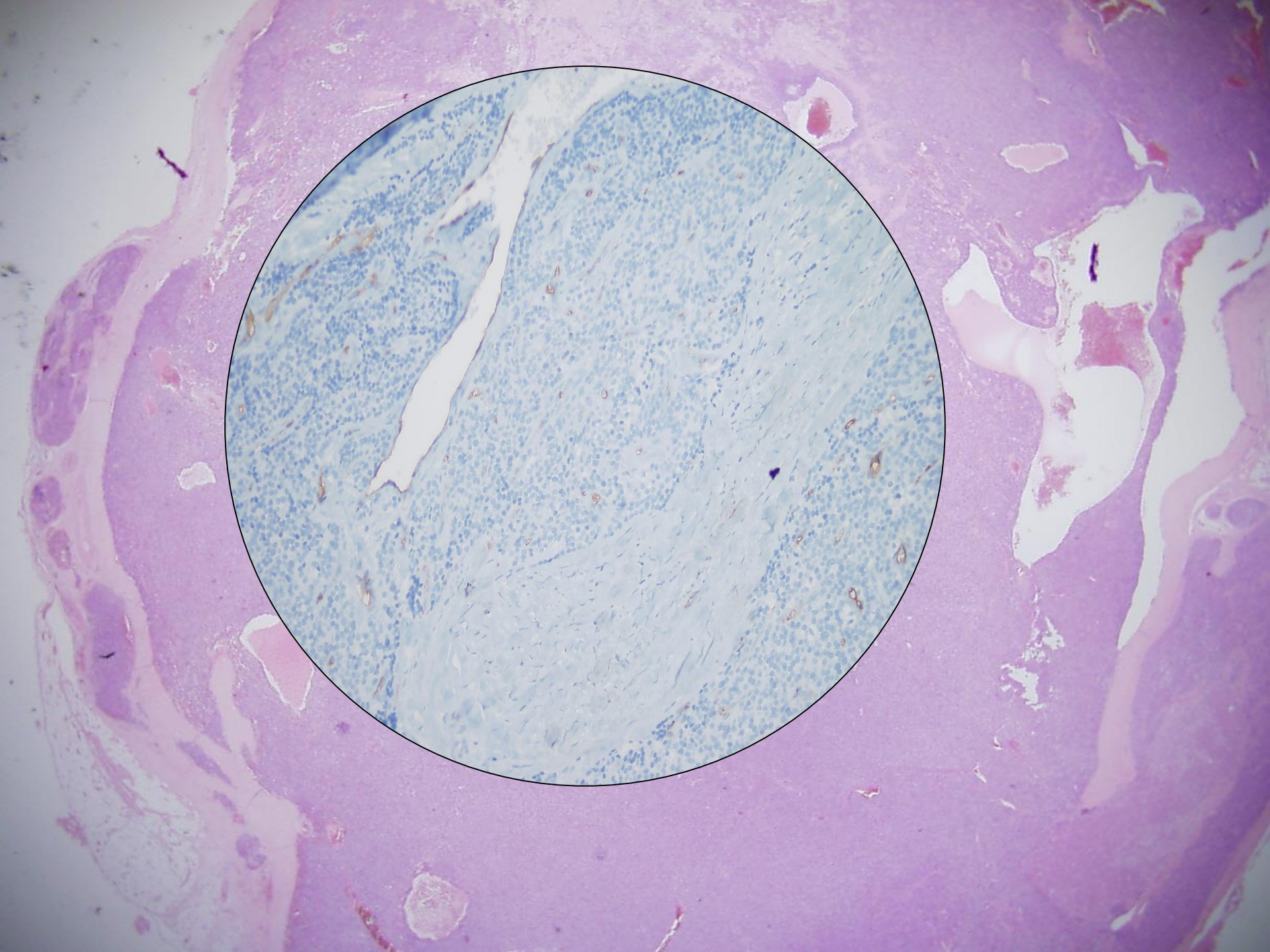









Secondary hyperparathyreosis

Associated by renal insufficiency

Lowered Calcium intake

Steatorrhea

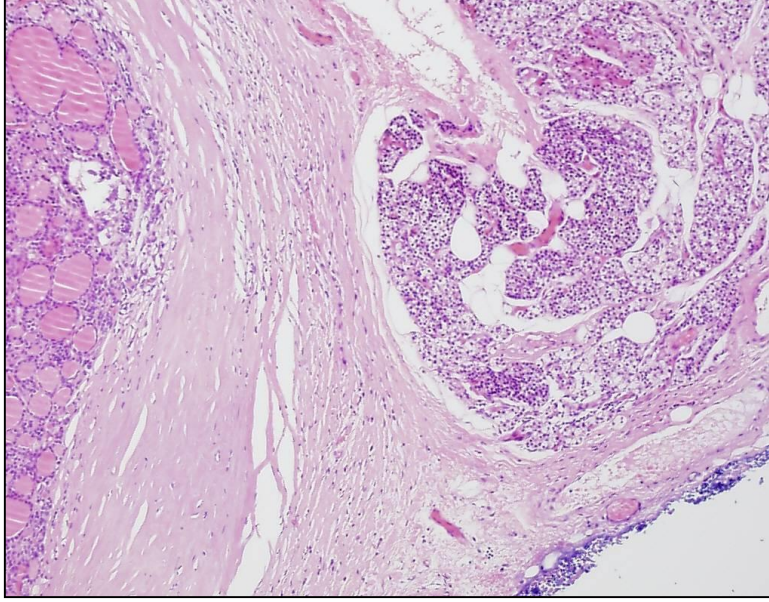
D vitamine deficiency

Low se Ca

Symptoms are similar to primary ~, but less severe

Tertiary hyperparathyreosis

Hypoparathyreosis



Surgical removal :(by accident)

Familiary ~ : chr mucocutan candidiasis, with primary adrenal failure (autoimmun polyendocrin sy – APS1)

Congenital aplasia, with (thymic aplasia, cardiac abnormalities)

Idiopathic ~ : caused by autoantibodies, that block the calcium sensing receptors(CASR) (no parat-hormone release)

Hypoparathyreosis

Tetania
- neuromuscular irritability
  Chovstek
  Trousseau's sign

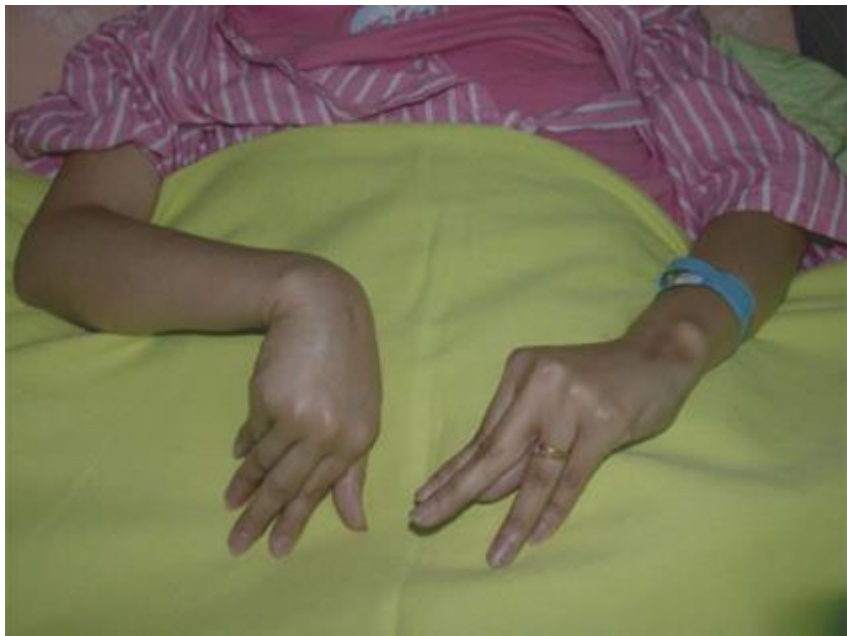
Mental alterations
  depression, irritability, hallucinations, psychosis

CNS
  basal ganglion calcification
  parkinson like signs
  papilla oedema

Lense calcification – cataracta

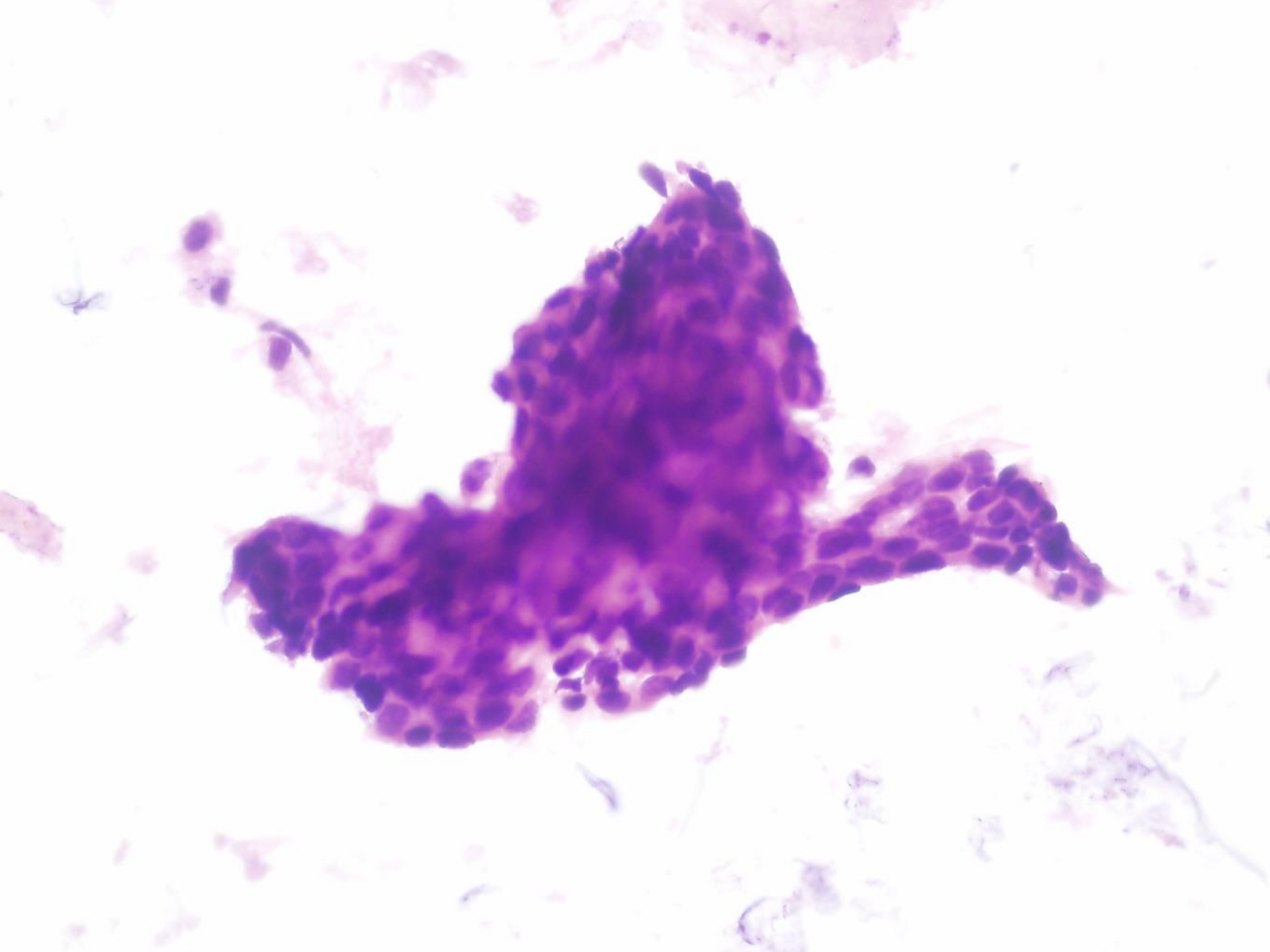
EKG – prolongation of QT interval

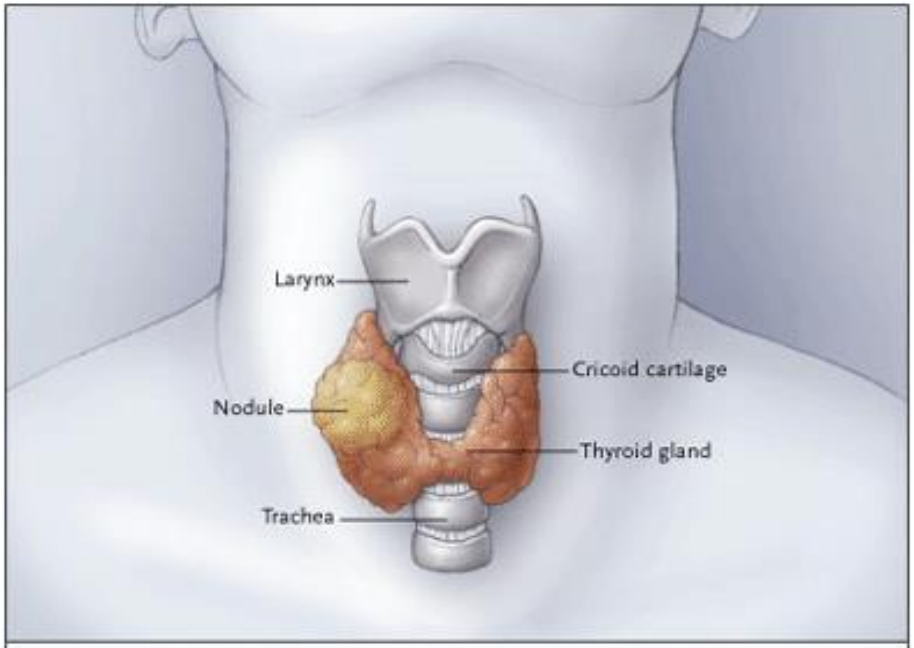
Dental abnormalities



Pseudohypoparathyreosis

End-organ resistance to PTH





Benign Nodules (95%)	Carcinomas (5%)
Hyperplastic nodules (85%)	Papillary (81%)
Adenomas (15%)	Follicular and Hürthle-cell (14%)
Cysts (<1%)	Medullary (3%)
	Anaplastic (2%)

- **Circulation**
- Most of the thyroid hormone circulating in the blood is bound to transport proteins. Only a very small fraction of the circulating hormone is free (unbound) and biologically active, hence measuring concentrations of free thyroid hormones is of great diagnostic value.
- When thyroid hormone is bound, it is not active, so the amount of free T3/T4 is what is important. For this reason, measuring total thyroxine in the blood can be misleading.

- The **thyroid hormones**, thyroxine (**T4**) and triiodothyronine (**T3**), are tyrosine-based hormones produced by the thyroid gland. An important component in the synthesis is iodine. The major form of thyroid hormone in the blood is thyroxine (T4). The ratio of T4 to T3 released in the blood is roughly 20 to 1. Thyroxine is converted to the active T3 (three to four times more potent than T4) within cells by deiodinases (5'-iodinase). These are further processed by decarboxylation and deiodination to produce iodothyronamine (**T1a**) and thyronamine (**T0a**).

- T3 and T4 cross the cell membrane, probably via amino acid importins, and function via a well-studied set of nuclear receptors in the nucleus of the cell, the thyroid hormone receptors.
- T1a and T0a are positively charged and do not cross the membrane; they are believed to function via the trace amine-associated receptor *TAAR1* (TAR1, TA1), a G-protein-coupled receptor located in the cell membrane.
- Another critical diagnostic tool is the amount of thyroid-stimulating hormone (TSH) that is present.

- **Function**
- The thyronines act on the body to increase the basal metabolic rate, affect protein synthesis and increase the body's sensitivity to catecholamines (such as adrenaline) by permissiveness. The thyroid hormones are essential to proper development and differentiation of all cells of the human body. These hormones also regulate protein, fat, and carbohydrate metabolism, affecting how human cells use energetic compounds. Numerous physiological and pathological stimuli influence thyroid hormone synthesis.
- The thyronamines function via some unknown mechanism to inhibit neuronal activity; this plays an important role in the hibernation cycles of mammals and the moulting behaviour of birds. One effect of administering the thyronamines is a severe drop in body temperature.

- **Summary of the effects of hormones on skeletal metabolism**
- **Increase Bone resorption**
- Parathyroid hormone
  Glucocorticoids
  Thyroid Hormone
  Vitamin D metabolites in high doses **Decrease Bone Resorption**
- Calcitonin
  Gonadal steroids **Increase Bone Formation**
- Growth hormone
  Vitamin D metabolites
  Gonadal steroids **Decrease Bone Formation**
- Glucocorticoids

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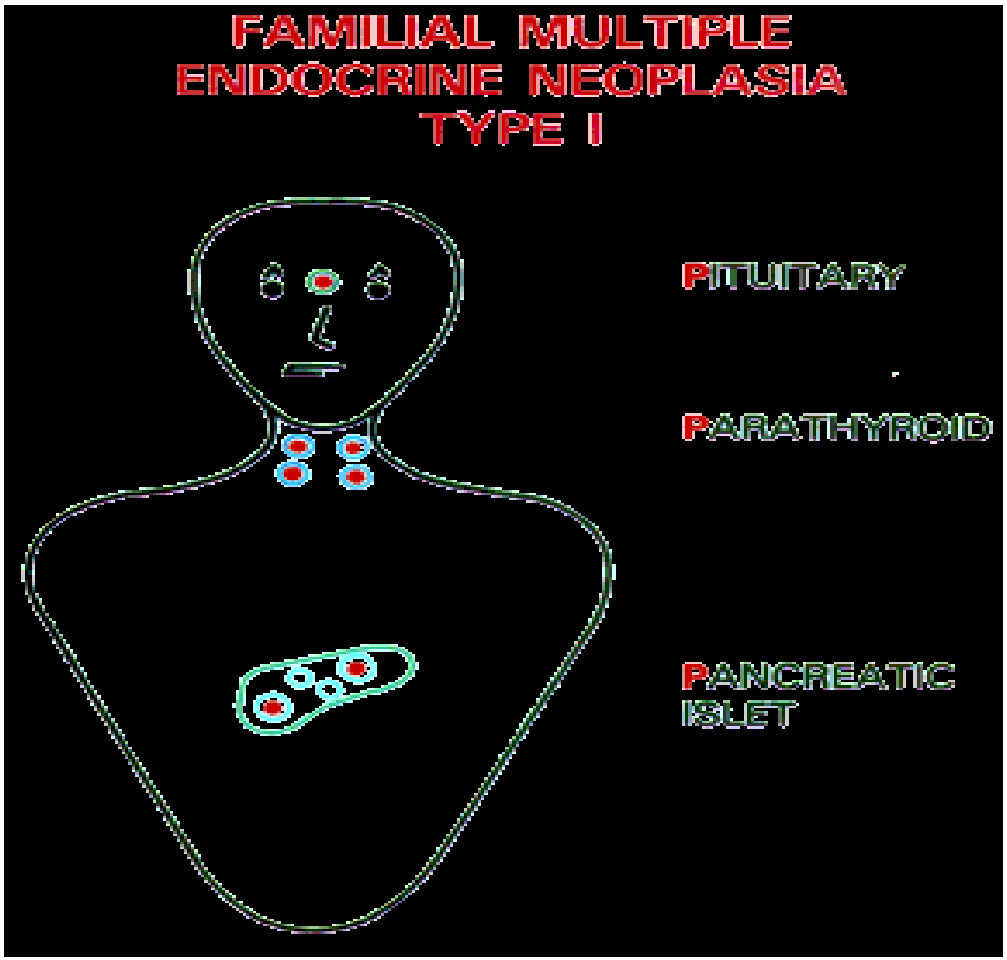
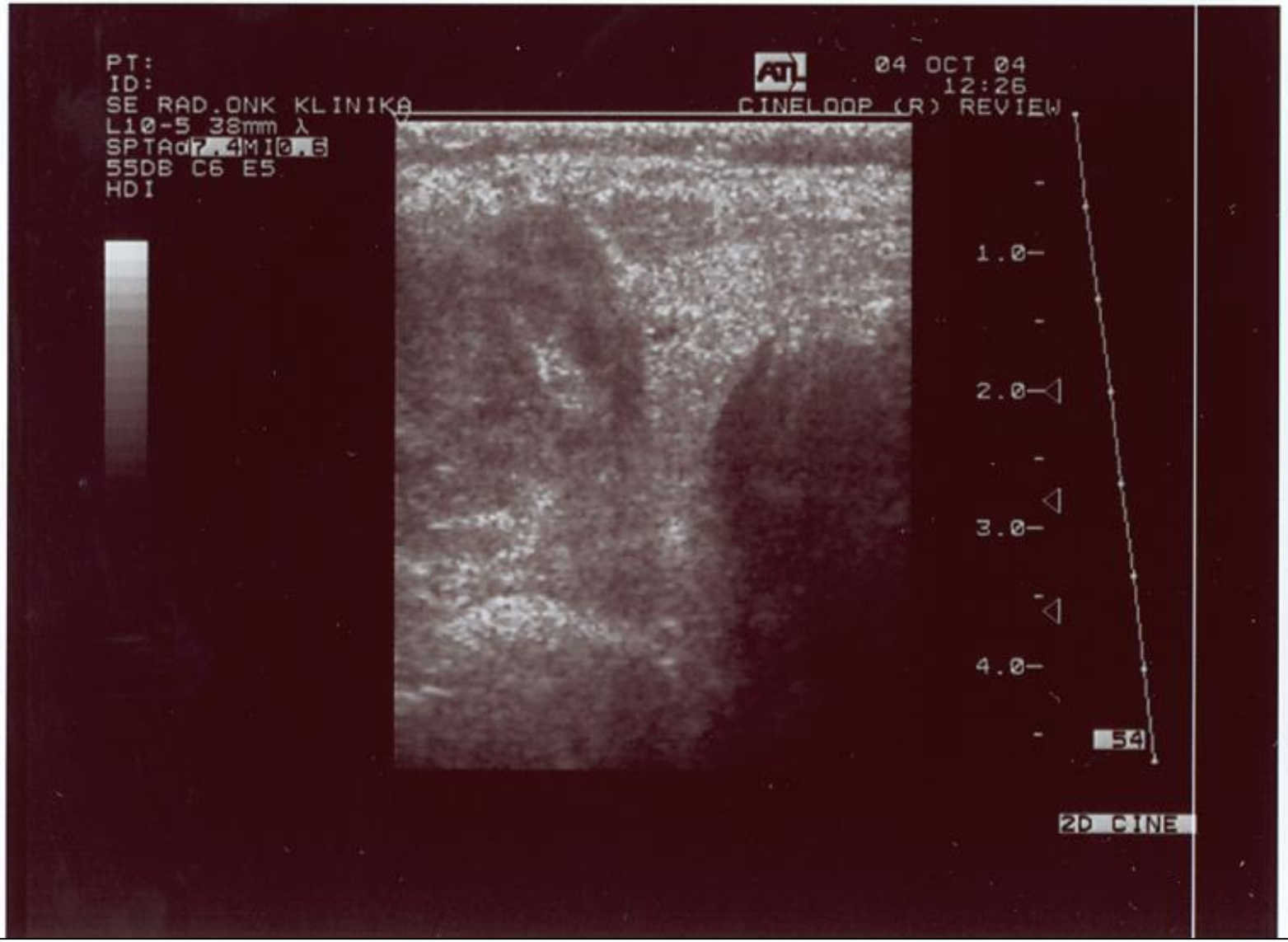
FAMILIAL MULTIPLE ENDOCRINE NEOPLASIA TYPE I

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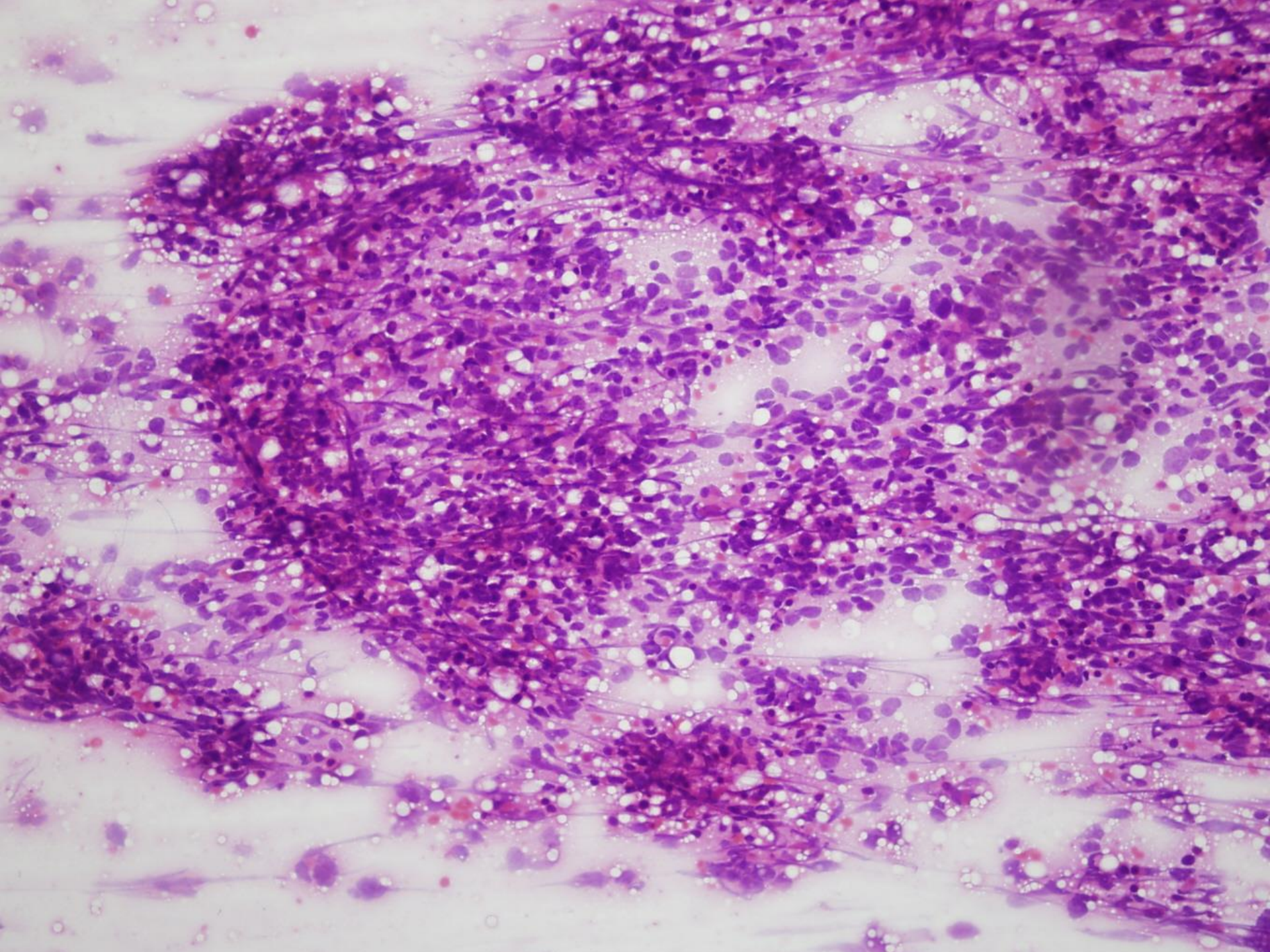
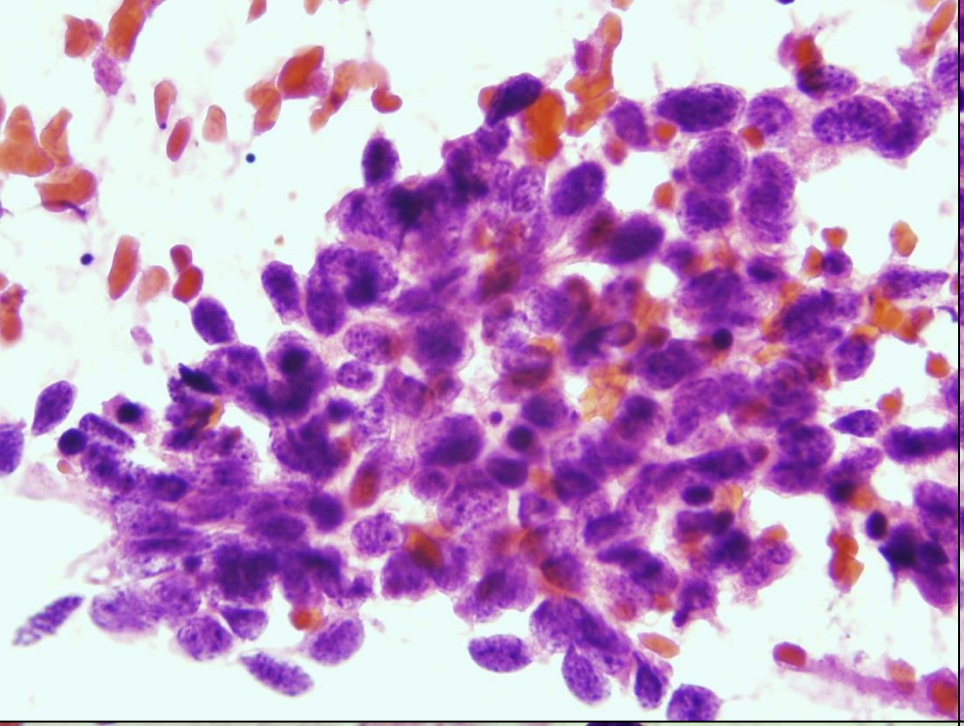
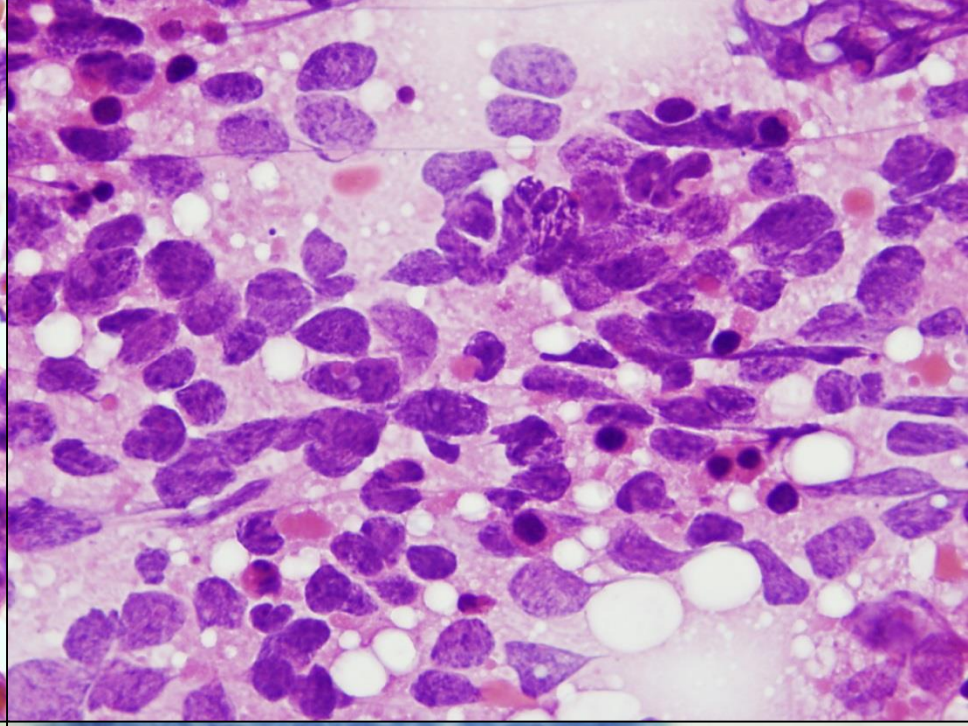
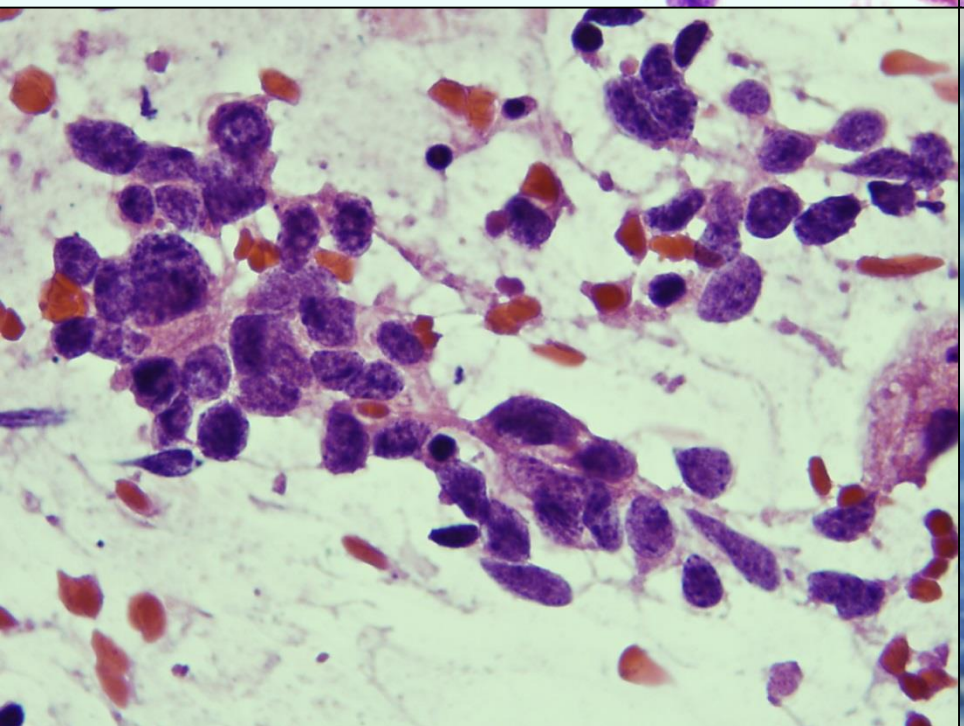
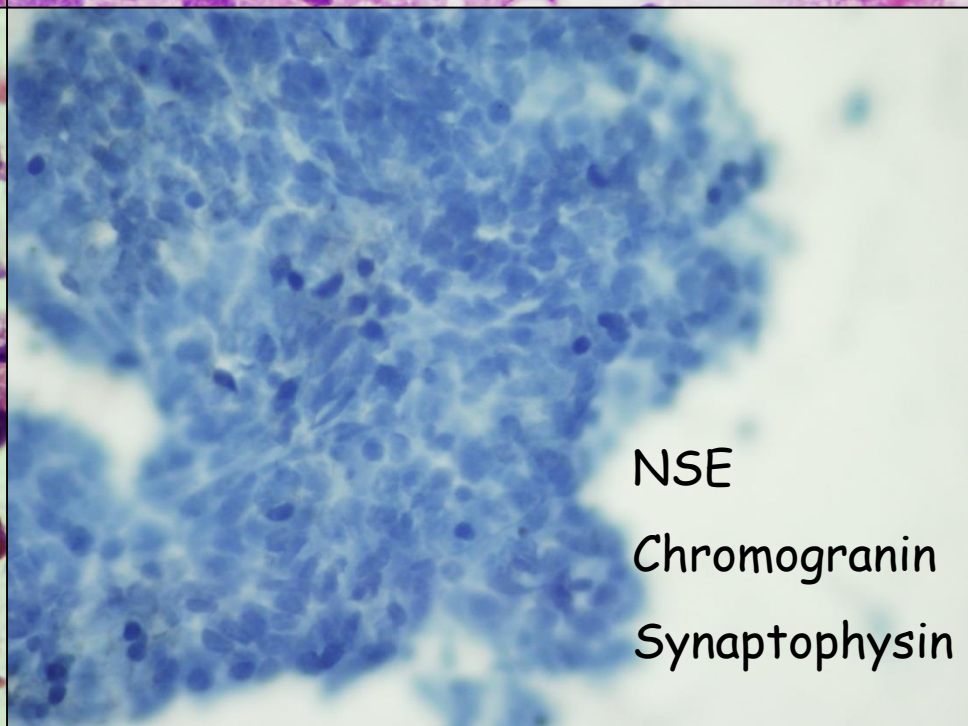
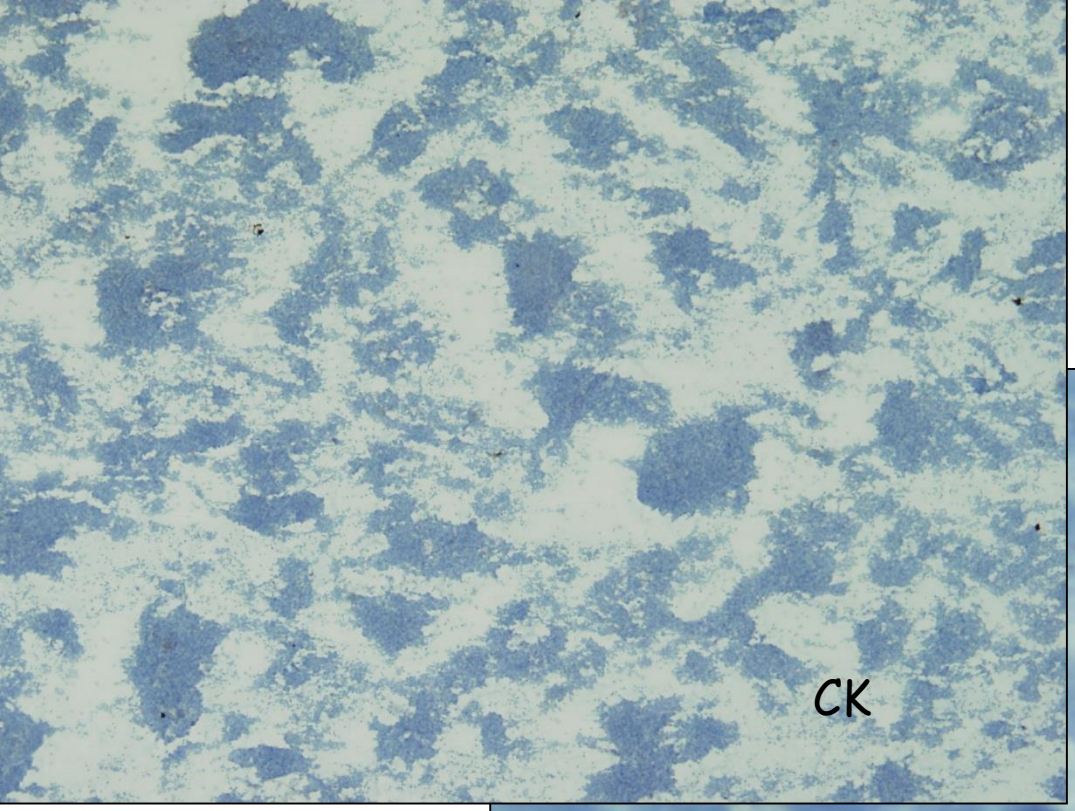









NSE
Chromogranin
Synaptophysin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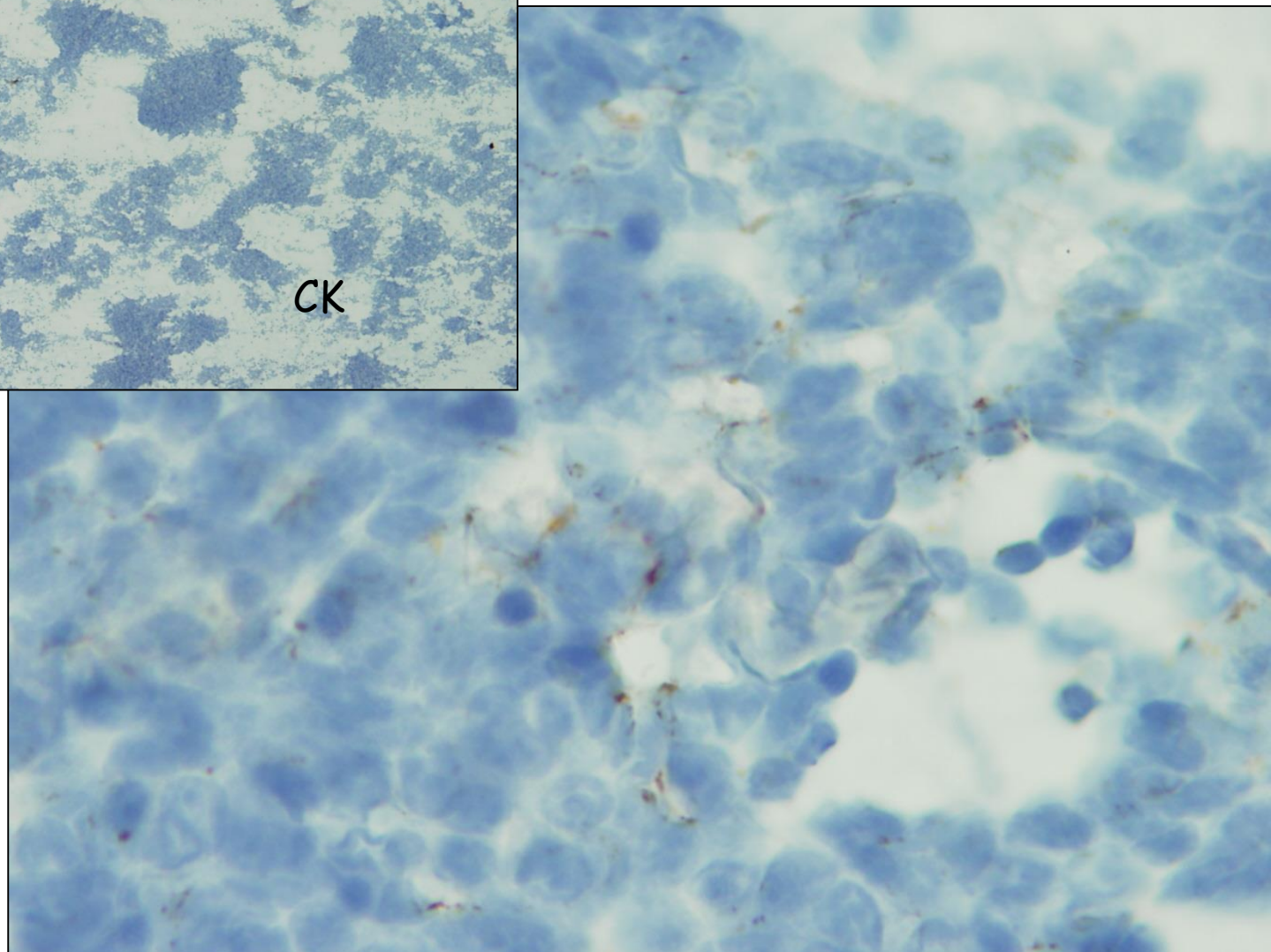

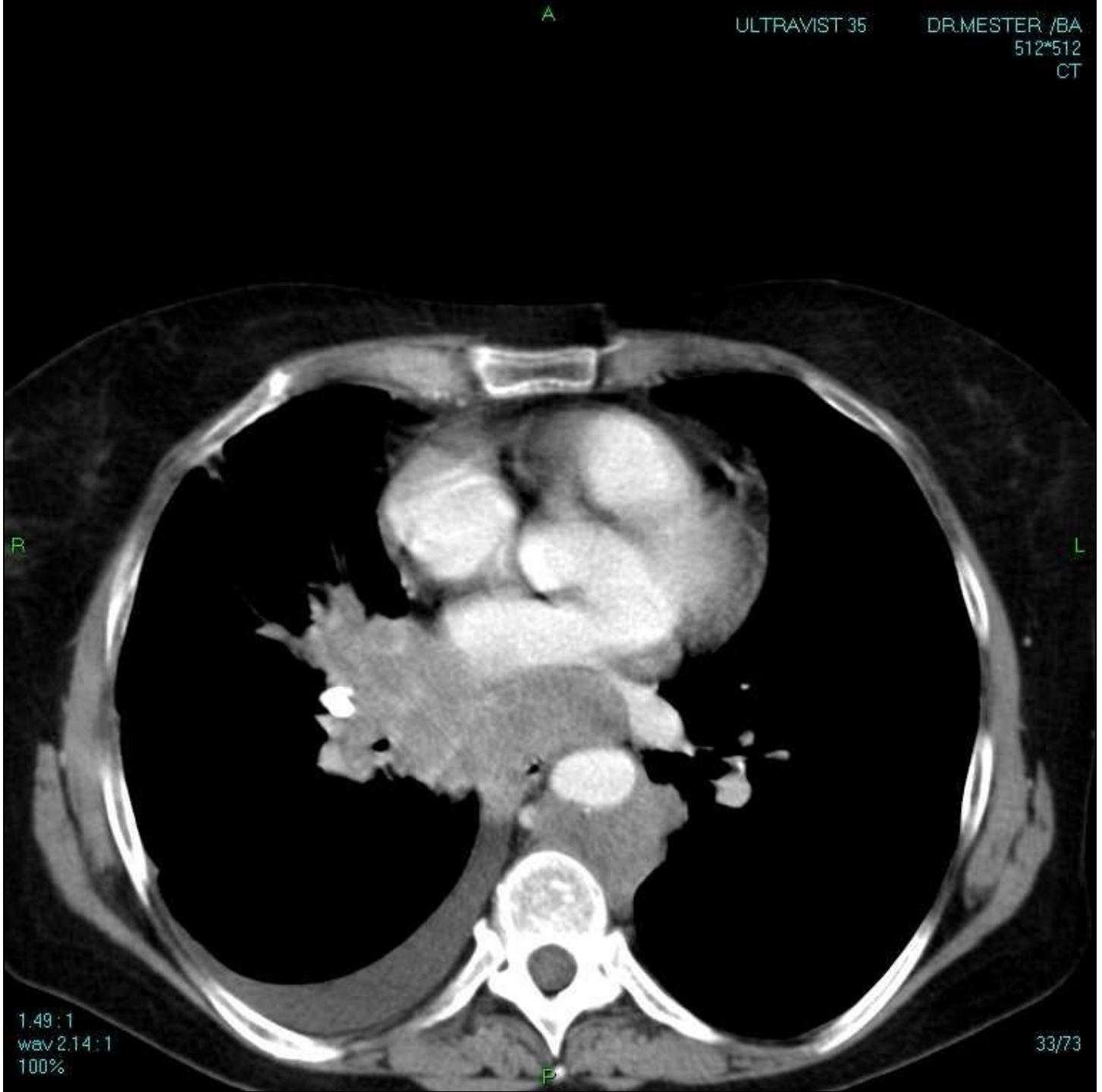
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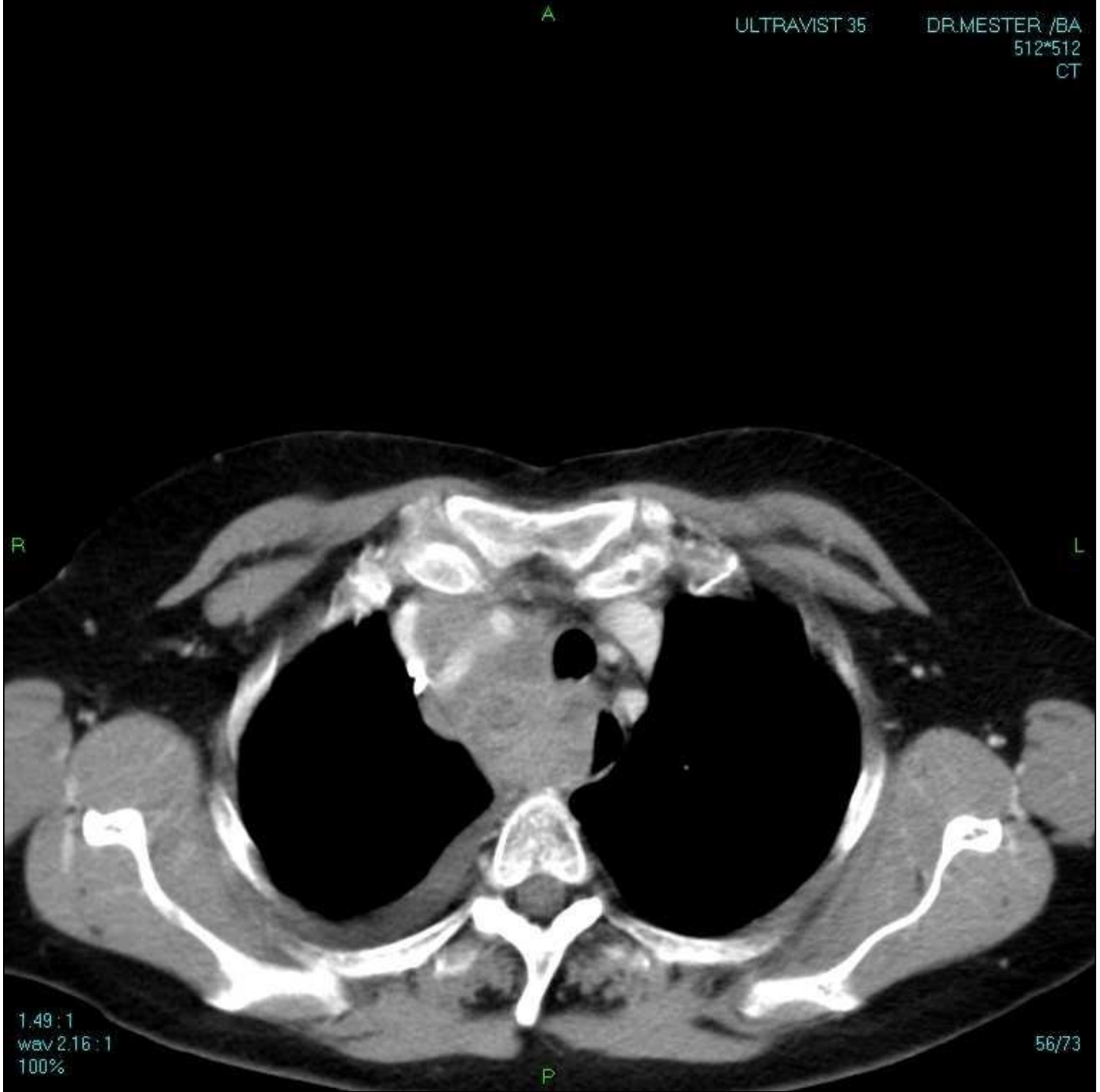
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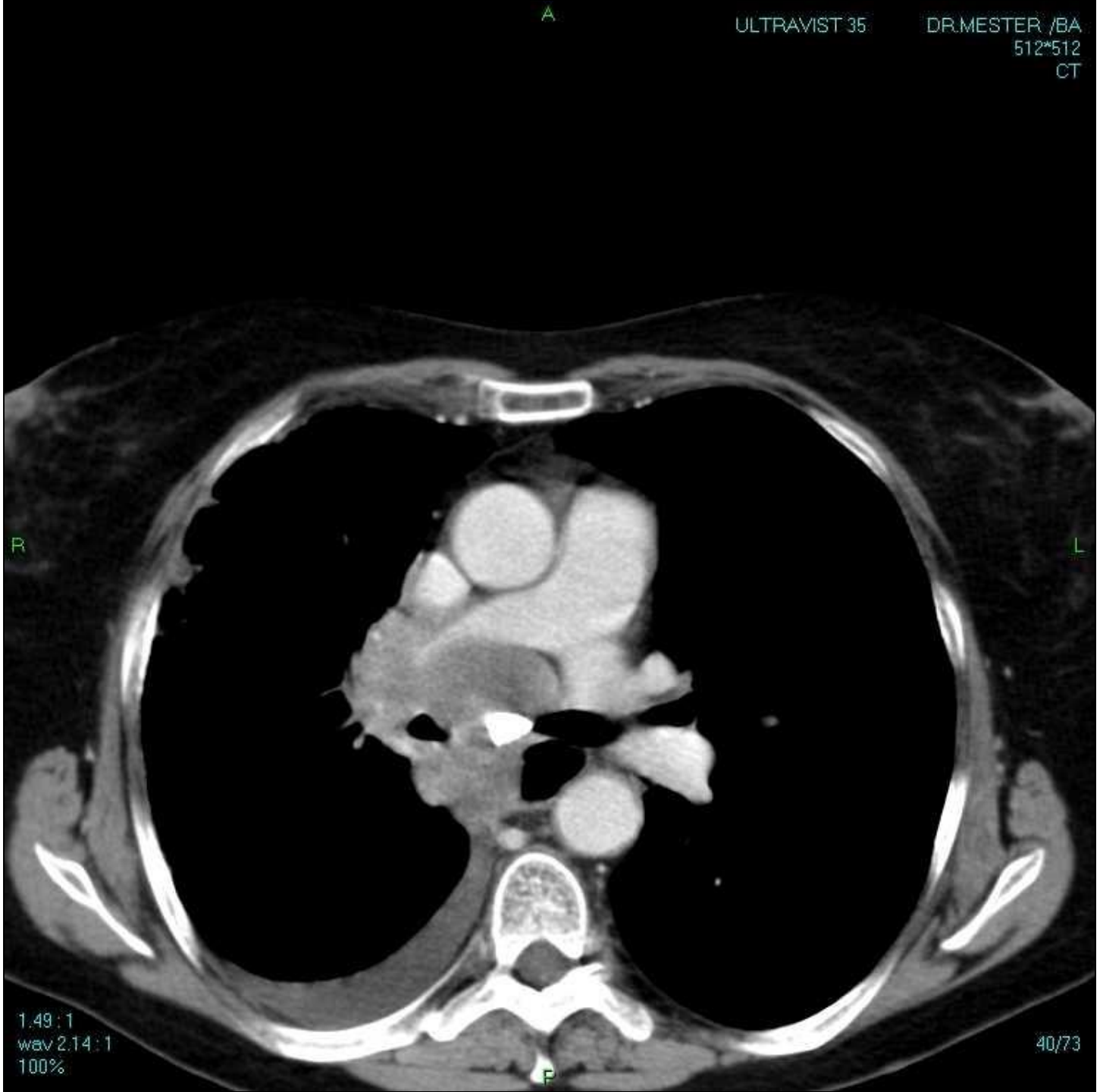
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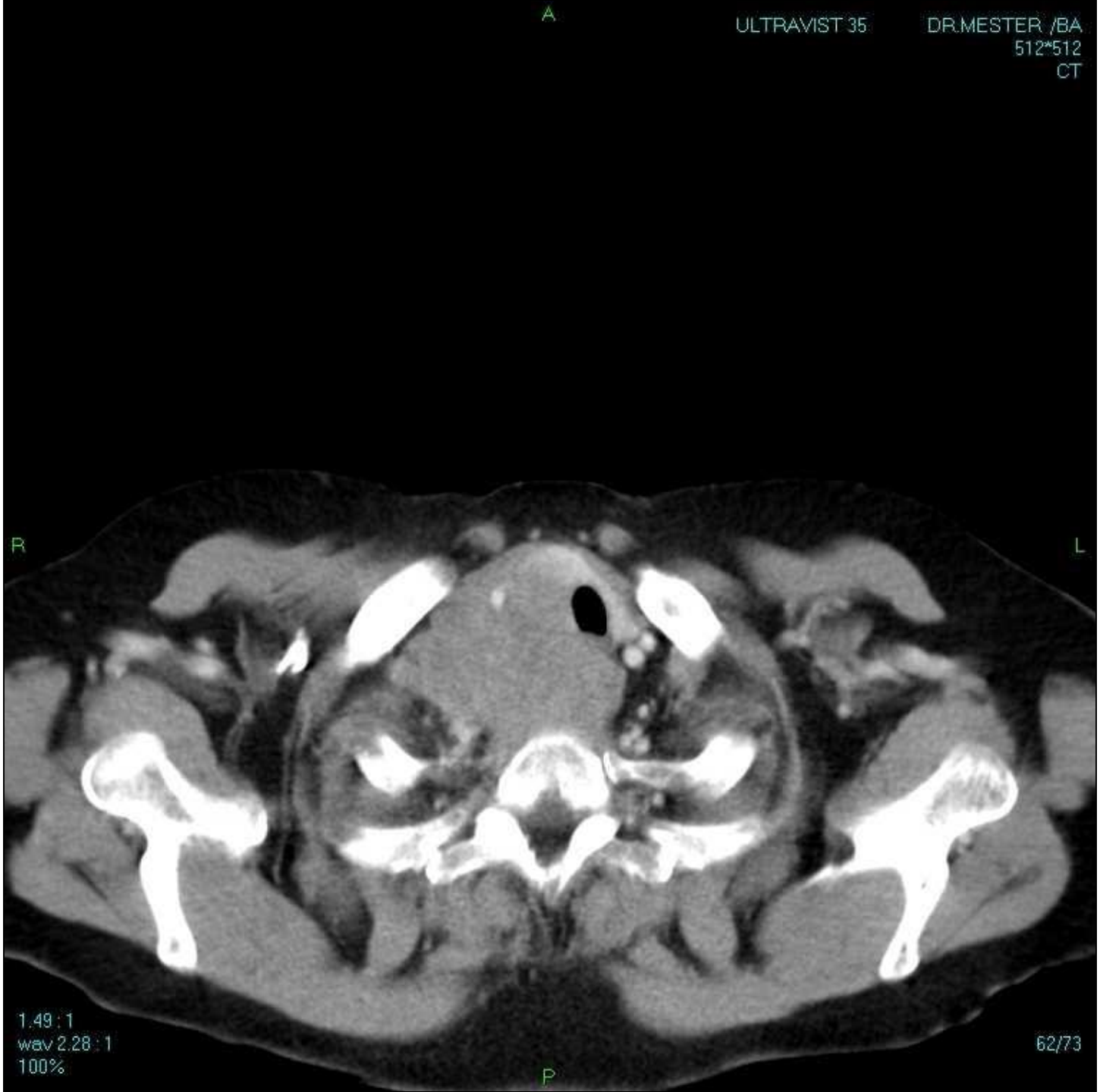
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