a group of symptoms, mainly AG hyperfun (hypercortisolism) -it can be caused by lots of things, mostly cushing disease intake of GC - escpecially latrogenics (most causer of CS) Cushing disease	Cushing syndrome					
Exogenous intake of GC - escpecially iatrogenics (most causer of CS) Cushing disease -Mostly (mostly in females) -PG tumors ectopic corticotropins syndrome -ACTH-secreting pulmonary small-cell carcinoma AG tumors Adenoma (mostly - Fe) or carcinoma Macronodular hyperplasia ectopic expression of the receptors: GIPR, LHR, serotonin & ADH Primary pigmented nodular AG Genes: PRKARIA & PDE11 McCune Albright syndrome Genes: GNAS	ic					
Etiology Endogenous ACTH dependent ACTH independent ACTH-dependent only) -nodular hyperplasia (with ACTH-dependent only) -nodular hyperplasia (macro >3cm - micro <3cm) -tumors (adenoma or carcinoma) -central obesity -facial plethora (erythromatous) -hypo libido -menstrual disturbance -hypertension -females hirsutism -depression -glc intolerance -proxima muscles weakness -thin extremities -osteopenia (easy fractures) -thin extremities -osteopenia (easy fractures) -thin skin (easy bruises & bleeding) -dec children linear growth -nephrolithiasis (renal stones) -immunosuppression -abdominal stria (due to collagen def & small vessles rupture) Serum ACTH -low →primary CS (AG path) -high →secondry CS (PG tumors or ectopic) High DXM -ACTH suppression → PG tumor	15	-it can be caused by lots of things, mostly cushing disease				
Can only be 1 of the following: -cortical atrophy (due to exogenous GC) -hyperplasia (macro >3cm - micro <3cm) -tumors (adenoma or carcinoma) -central obesity -moon face -menstrual disturbance -females hirsutism -glc intolerance -females hirsutism -nephrolithiasis (renal stones) -influences -finel service or ticotropins syndrome -ACTH-secreting pulmonary small-cell carcinoma -AG tumors -Adenoma (mostly - Fe) or carcinoma -AG tumors -Adenoma (mostly - Fe) or carcinoma -AG tumors -Adenoma (mostly - Fe) or carcinoma -Macronodular hyperplasia -ectopic expression of the receptors: GIPR, LHR, serotonin & ADH -primary pigmented nodular AG -Genes: PRKARIA & PDE11 -McCune Albright syndrome -Genes: GNAS -micro <3cm) -tumors (adenoma or carcinoma) -central obesity -facial plethora (erythromatous) -hypo libido -hypertension -depression -glc intolerance -proxima muscles weakness -thin extremities -osteopenia (easy fractures) -thin skin (easy bruises & bleeding) -dec children linear growth -nephrolithiasis (renal stones) -immunosuppression -abdominal stria (due to collagen def & small vessles rupture) -low → primary CS (AG path) -high → secondry CS (PG tumors or ectopic) -ACTH suppression → PG tumor		Fyogenous	intake of GC -	escpecially iatrogenics		
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Diagnosis -high →secondry CS (PG tumors or ectopic) High DXM -ACTH suppression → PG tumor	Diagnosis		<u> </u>			
High DXM -ACTH suppression → PG tumor						
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Hyperaldosteronism				
ls	Multiple or one cause that results in excessive aldosterone			
	Primary	Conn syndrome -AG pathy -Ai causing: hypersecretion of aldosterone &		
Etiology	Secondry	-it could be familial (but rare) Pathies -nephrosclerosis: results in renal hypofun (its due to renal artery stenosis) -hypovolemia: ass. With edemia (its due to HF, nephrotic syndrome, liver cirrhosis) -preg: (estrogen causes renin hyperfun) signs -hypersecretion of aldosterone -hyperfun of renin-angiotensin system		
Symptms	-secondry hypertension (left ventricle hypertrophy, MI & strokes			

AG hypofun					
AKA	Adrenal insuff.				
	Primary	Acute	-AKA: adrenal crisis		
	(AG disease)		-waterhouse-friderichsen syndrome		
			can be cong.		
			-Sudden CorticoS. Withdrawal after		
			prolonged treatment		
			-Stresses in pt with underlying chronic		
Etiology			AG hypofun		
		chronic	-Addison disease		
			-Fungal inf -Hemochromatosis		
			-Sarcoidsosis -Systemic amyloidosis		
			-Ai -Tb -Tumor metastases		
	Secondry	-caused l	by PG disease		
	(insuff ACTH)				
	-AG intra-hemorrhage resulting in bilateral AG failure				
waterhouse-	, ,	•	B: meningococcus neisseria meningitis)		
friderichsen			son (targets kids)		
	-significantly high fever & skin rash (hemorrhages)				
adrenal	-fatigue				
crisis	-dehydration				
symptoms	-drop in BP (vascular collapse)				
-renal shut down (hypoNatremia & hyperkalemia) Addison disease					
Aka	Hyperaldoster		in disease		
7 110	Military TB rea		<u> </u>		
	-systemic spread of TB				
	Septicemia				
	-waterhouse-friderichsen				
Etiology	Ai				
	-targets Z.glomerulosa				
	-T-cells is the main destructor				
	-mostly pt would have other underlying Ai diseases				
	-skin pigmentation (around: cheeks, forhead, creases & scars -				
	we think that its caused by indirect act of ACTH on melanocytes)				
Symptoms	-electrolytes imbalance (hypoNatremia & hyperkalemia)				
	-lethargy (fatigue)				
	-hypotension				

Pleochromocytoma PCM				
Is	Tumor of chromaffin cells of AGM			
Etiology	The rule of 10%s -10% of non-familial PCM are bilateral (70% if its familial) -10% of PCM is familial related (MEN2-A/B gene) -10% of PCM arise in extra-AG sites (bladder) -10% of PCM are malignant -10% of PCM target children			
Symptoms	-CAT hypersecretion (cuz chromaffin cells secrete them normally) -hypertension (surgically correctable - like aldosterone-secreting tumor)			
Diagnosis	-we look for CAT and their products in serum & urine (metanE & vanillyimandelic acid)			
Treatment	Tumor excision			
MEN2-A	-thyroid medullary carcinoma -C-cells hyperplasia -PCM			
mutation	-parathyroid hyperplasia			
MEN2-B mutation	-thyroid medullary carcinoma -C-cells hyperplasia -PCM -mucosal neuroma -marfonoid (skeleton mutations)			

Other			
	-its features are non-like other of any carcinoma, its really hard to		
AGC	diagnose, so we rely on the weight of the AG, if it increased		
carcinoma	,		
	-hyperplasia & invasion are the main to risks		
	-ass with: inc androgens & dec cortisol/aldosterone		
	-caused by enz def (21/11-hydroxylase), the enz usually breaks		
	estrogens & progestorones, so when its def they'll be highly		
Cong. AG	elevated		
hyperplasia	-causes kids ambiguous genitalia		
	-if the enz is only partially mutated, female would have		
	pre-puberty, hirsutism & voice hoarsness		
	-neonatal screening for the enz is essential		
	-fibromascı	ular hyperplastic renal artery	
Hypertension	(stenosis - d	detectable by angiography)	
rare causes	-polycystic kidney (autosomal recessive - abdominal mass)		
Tale Causes	-Conn syndrome (check for electrolytes balance)		
	-pheochromocytoma (check for CAT in serum & urine)		
	Signs		
	symptoms	-sking café au lait spots	
		-hemorrhages	
		-	
	-Von recklir	nghausen neurofibromastosis type1	
	-café au lat skin spots		
	-schwannoma		
	-meningioma		
\/DNIN/11	-glioma		
VRNM1	-PCM		
	-hemorrhages		
	-well-defined tumor (polygonal or spindle chromaffin or chief cells)		
	-sustentacular small cell		
	-zellballen nests		