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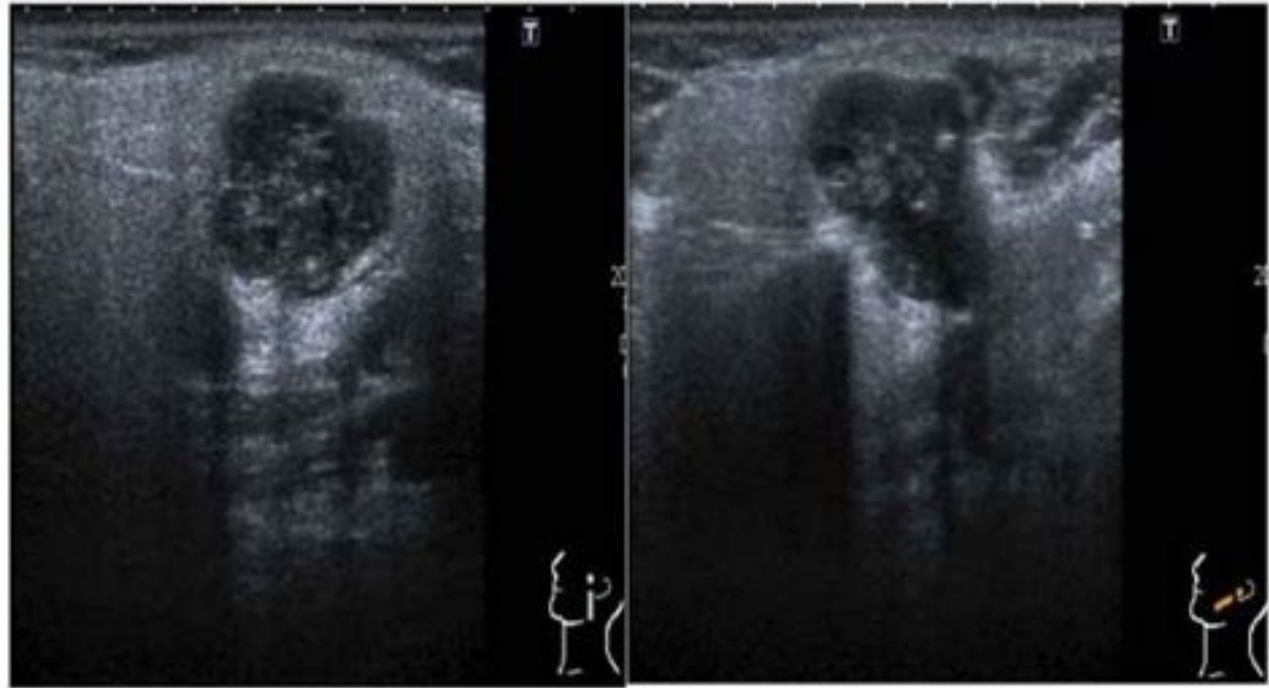
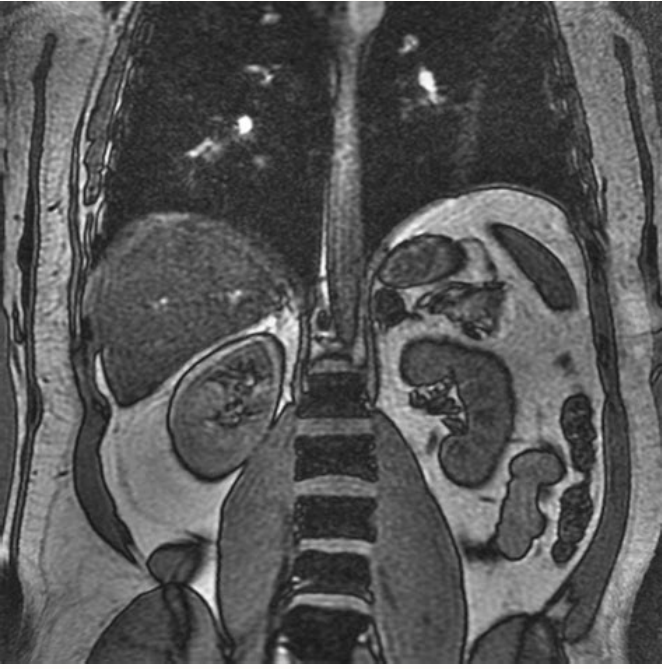


Figura 2. Corte ecográfico longitudinal y transversal de la glándula parótida izquierda

Adenoma Suprarrenal

- Aspecto inespecífico.
- Tamaño menor a 5 cm.
- Contornos lisos y definidos.
- Sin hemorragia ni necrosis.
- Mínima captación de contraste (<30 UH), con wash out >50%.
- Calcificaciones infrecuentes.
- Valores de atenuación entre 0 y 10 UH (contenido graso).



Adenoma suprarrenal radiopaedia. Adenoma suprarrenal izquierdo. Adenoma suprarrenal tratamiento. Adenoma suprarrenal pathology outlines. Adenoma suprarrenal sintomas. Adenoma suprarrenal que es. Adenoma suprarrenal derecho. Adenoma suprarrenal pdf.

Este artículo o sección necesita referencias que aparezcan en una publicación acreditada.Este aviso fue puesto el 11 de marzo de 2014. Busca fuentes: «Adenoma» – noticias · libros · académico · imágenes Adenoma Adenoma de colonEspecialidad oncología Aviso médico [editar datos en Wikidata] Un adenoma (del griego ἀδέν, adén, «glándula») es un tumor epitelial benigno cuya estructura interna es semejante a la de una glándula.[1][2] Los adenomas pueden crecer a partir de muchos órganos glandulares, incluidas las glándulas suprarrenales, la glándula pituitaria, la tiroides, la próstata y otras. Algunos adenomas crecen a partir del tejido epitelial en áreas no glandulares, pero expresan la estructura del tejido glandular (como puede suceder en la poliposis adenomatosa familiar). Aunque los adenomas son benignos, con el tiempo pueden transformarse y volverse malignos.[3] en cuyo caso se denominan adenocarcinomas. La mayoría de los adenomas no se transforman.[4] Pero, incluso cuando son benignos, tienen el potencial de causar graves complicaciones de salud al comprimir otras estructuras[5] (efecto de masa) y al producir grandes cantidades de hormonas de una manera no regulada, no dependiente de la retroalimentación (causando síndromes paraneoplásicos). Algunos adenomas son demasiado pequeños para ser vistos macroscópicamente, pero aún pueden causar síntomas clínicos. Tipos de adenoma Existen muchas clases diferentes de adenomas según la localización, como:[6] Adenoma tiroideo: aparece en la glándula tiroides. También se llama nódulo tiroideo. Puede ser foliular o papilar, funcionante o no funcionante. Cuando es un adenoma hiperfuncionante (adenoma de Plummer) se llama adenoma tóxico y produce hipertiroidismo. Adenoma suprarrenal: aparece en la glándula suprarrenal. También se llama adenoma adrenal. La mayoría son no funcionantes o incidentalmos puesto que se descubren en un TAC realizado por otro motivo. Cuando son funcionantes pueden producir cortisol, aldosterona (síndrome de Conn), andrógenos o catecolaminas (feocromocitoma). Adenoma bronquial: aparece en los bronquios. También se llama adenoma de Jackson. Muchos son tumores carcinoides. Adenoma de colon: aparece en el colon. Adquieren en la mayoría de los casos la morfología de pólipos de colon. Adenoma de próstata: aparece en la próstata. También se llama hiperplasia benigna de próstata.. Adenoma pleomórfico: aparece frecuentemente en las glándulas salivares.[7] Adenoma hepático: aparece en el hígado.[8][9] Adenoma hipofisario: aparece en la hipófisis. La mayoría no son funcionantes no producen y no secretan hormonas; entre los productores de hormonas los más frecuentes son los prolactinomas, asociados con una elevada secreción de prolactina; si sintetizan hormona del crecimiento provocan gigantismo o acromegalia; si producen ACTH se desarrolla la enfermedad de Cushing, la variante hipofisaria del Síndrome de Cushing. Adenoma de mama: se llaman fibroadenomas. Frecuentemente son muy pequeños, y difíciles de detectar. Suelen no dar síntomas. El tratamiento puede incluir biopsia por agua, o remoción. Adenoma de páncreas: puede ser cualquier tumor benigno de páncreas. Cuando es funcional se lo denomina insulinoma porque produce insulina, gastrinoma si produce gastrina, vipoma si produce Péptido vasoactivo intestinal. Adenoma paratiroideo: está localizado en las glándulas paratiroides. [10] Es la causa más frecuente de hiperparatiroidismo por exceso de producción de hormona paratiroidea Adenoma testicular o adenoma de Pick, localizado en los testículos, en las células de Sertoli. Aparece con frecuencia en los riñones. Véase también Papioma Referencias 1 Melloni, Biagio John; Dox, Ida; Eisner, Gilbert M. (1982). Diccionario médico ilustrado de Melloni. Reverte. ISBN 9788429155488. Consultado el 4 de febrero de 2018. ↑ ◄ www.cancer.gov. 2 de febrero de 2011. Consultado el 3 de diciembre de 2021. ↑ Benet i Batista-Alentorn, Josep M. (1969). Gran Enciclopèdia Catalana (en catalán) 1. Barcelona: Enciclopèdia Catalana, p. 165. D.L. B 42775 1968. Consultado el 28 de abril de 2020. ↑ Zúñiga S.R. (1973). «Adenocarcinoma del Colon y Recto». *Rev. Med. Hondur.* 41: 63. |fechaacceso= requiere |url= (ayuda) ↑ Nueva Enciclopedia Larousse 1 (2a edición). Barcelona : Madrid: Planeta, 1984. p. 114. ISBN 84-320-4241-2. ↑ Cumulated Index Medicus (en inglés). 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ISSN 1936-0541. PMC 2716879. PMID 19669260. doi:10.1007/s12072-008-9075-0. Consultado el 3 de diciembre de 2021. ↑ «MedLine Plus. Adenomas paratiroideos». Datos: Q272741 Multimedia: Adenomas Obtenido de « This article is about medically-recognized chronic adrenal insufficiency. For a term used in alternative medicine, see Adrenal fatigue. Medical conditionAdrenal insufficiencyOther namesAdrenocortical insufficiency, hypocorticism, hypocortisolism, hypoadrenalismAdrenal glandSpecialtyEndocrinology Adrenal insufficiency is a condition in which the adrenal glands do not produce adequate amounts of steroid hormones, primarily cortisol, but may also include impaired production of aldosterone (a mineralocorticoid), which regulates sodium conservation, potassium secretion, and water retention.[1][2] Craving for salt or salty foods due to the urinary losses of sodium is common.[1][2] Causes can include brain tumors and sudden withdrawal from long-term exogenous steroid use (which is the most common cause overall).[10] Signs and symptoms Signs and symptoms include: hypoglycemia, hyperpigmentation, dehydration, weight loss, and disorientation. Additional signs and symptoms include weakness, tiredness, dizziness, low blood pressure that falls further when standing (orthostatic hypotension), cardiovascular collapse, muscle aches, nausea, vomiting, and diarrhea. These problems may develop gradually and insidiously. Addison's disease can present with tanning of the skin that may be patchy or even all over the body. Characteristic sites of tanning are skin creases (e.g. of the hands) and the inside of the cheek (buccal mucosa). Goitre and vitiligo may also be present.[4] Eosinophilia may also occur.[11] Hyponatremia is a sign of secondary insufficiency.[12] Causes Causes of acute adrenal insufficiency are mainly sudden withdrawal of long-term corticosteroid therapy, Waterhouse-Friderichsen syndrome, and stress in people with underlying chronic adrenal insufficiency.[13] The latter is termed critical illness-related corticosteroid insufficiency.[citation needed] For chronic adrenal insufficiency, the major contributors are autoimmune adrenalitis (Addison's Disease), tuberculosis, AIDS, and metastatic disease.[13] Minor causes of chronic adrenal insufficiency are systemic amyloidosis, fungal infections, hemochromatosis, and sarcoidosis.[13] Autoimmune adrenalitis may be part of 2 autoimmune polyglandular syndrome, which can include type 1 diabetes, hyperthyroidism, and autoimmune thyroid disease (also known as autoimmune thyroiditis, Hashimoto's thyroiditis, and Hashimoto's disease).[14] Hypogonadism may also present with this syndrome. Other diseases that are more common in people with autoimmune adrenalitis include premature ovarian failure, celiac disease, and autoimmune gastritis with pernicious anemia.[15] X-Linked Recessive Adrenoleukodystrophy can also cause adrenal insufficiency.[16] Adrenal insufficiency can be caused by the mechanism through which they cause the adrenal glands to produce insufficient cortisol. These are adrenal dysgenesis (the gland has not formed adequately during development), impaired steroidogenesis (the gland is present but is biochemically unable to produce cortisol) or adrenal destruction (disease processes leading to glandular damage).[17] Corticosteroid withdrawal Use of high-dose steroids for more than a week begins to produce suppression of the person's adrenal glands because the exogenous glucocorticoids suppress release of hypothalamic corticotropin-releasing hormone (CRH) and pituitary adrenocorticotrophic hormone (ACTH). With prolonged suppression, the adrenal glands atrophy (physically shrink), and can take months to recover full function after discontinuation of the exogenous glucocorticoid. During this recovery time, the person is vulnerable to adrenal insufficiency during times of stress, such as illness, due to both adrenal atrophy and suppression of CRH and ACTH release.[18][19] Use of steroids joint injections may also result in adrenal suppression after discontinuation.[20] Adrenal dysgenesis All causes in this category are genetic, and generally very rare. These include mutations to the SF1 transcription factor, congenital adrenal hypoplasia due to DAX-1 gene mutations and mutations to the ACTH receptor gene (or related genes, such as in the Triple A or Allgrove syndrome). DAX-1 mutations may cluster in a syndrome with glycerol kinase deficiency with a number of other symptoms when DAX-1 is deleted together with a number of other genes.[17] Impaired steroidogenesis To form cortisol, the adrenal gland requires cholesterol, which is then converted biochemically into steroid hormones. Interruptions in the delivery of cholesterol include Smith-Lemli-Opitz syndrome and abetalipoproteinemia. [verification needed] Of the synthesis problems, congenital adrenal hyperplasia is the most common (in various forms: 21-hydroxylase, 17α-hydroxylase, 11β-hydroxylase and 3β-hydroxysteroid dehydrogenase), lipid CAH due to deficiency of StAR and mitochondrial DNA mutations.[17] Some medications interfere with steroid synthesis enzymes (e.g. ketoconazole), while others accelerate the normal breakdown of hormones by the liver (e.g. rifampicin, phenytoin).[17] Adrenal destruction Autoimmune adrenalitis is the most common cause of Addison's disease in the industrialised world. Autoimmune destruction of the adrenal cortex is caused by an immune reaction against the enzyme 21-hydroxylase (a phenomenon first described in 1992).[21] This may be isolated or in the context of autoimmune polyendocrine syndrome (APS type 1 or 2), in which other hormone-producing organs, such as the thyroid and pancreas, may also be affected.[22] Adrenal destruction is also a feature of adrenoleukodystrophy (ALD), and when the adrenal glands are involved in metastasis (seeding of cancer cells from elsewhere in the body, especially lung), hemorrhage (e.g. in Waterhouse-Friderichsen syndrome or antiphospholipid syndrome), particular infections (tuberculosis, histoplasmosis, coccidioidomycosis), or the deposition of abnormal protein in amyloidosis.[23] Pathophysiology Hyponatremia can be caused by glucocorticoid deficiency. Low levels of glucocorticoids leads to systemic hypotension (one of the effects of cortisol is to increase peripheral resistance), which results in a decrease in stretch of the arterial baroreceptors of the carotid sinus and the aortic arch. This removes the tonic vagal and glossopharyngeal inhibition on the central release of ADH: high levels of ADH will ensue, which will subsequently lead to increase in water retention and hyponatremia.[citation needed] Differently from mineralocorticoid deficiency, glucocorticoid deficiency does not cause a negative sodium balance (in fact a positive sodium balance may occur).[24] Diagnosis The best diagnostic tool to confirm adrenal insufficiency is the ACTH stimulation test; however, if a patient is suspected to be experiencing an acute adrenal crisis, immediate treatment with IV corticosteroids is imperative and should not be delayed for any testing, as the patient's health can deteriorate rapidly and result in death without replacing the corticosteroids.[citation needed] Dexamethasone should be used as the corticosteroid if the plan is to do the ACTH stimulation test at a later time as it is the only corticosteroid that will not affect the test results.[25] If not performed during crisis, then labs to be run should include: random cortisol, serum ACTH, aldosterone, renin, potassium and sodium. A CT of the adrenal glands can be used to check for structural abnormalities of the adrenal glands. An MRI of the pituitary can be used to check for structural abnormalities of the pituitary. However, in order to check the functionality of the Hypothalamic Pituitary Adrenal (HPA) Axis the entire axis must be tested by way of ACTH stimulation test, CRH stimulation test and perhaps an Insulin Tolerance Test (ITT). In order to check for Addison's Disease, the auto-immune type of primary adrenal insufficiency, labs should be drawn to check 21-hydroxylase autoantibodies.[citation needed] Effects Source of pathology CRH ACTH DHEA DHEA-S cortisol aldosterone renin Na K Causes5 hypothalamus(tertiary)1 low low low low low3 low low low low the hypothalamus (adenoma), antibodies, environment (i.e. toxins), head injury pituitary(secondary) high2 low low low low3 normal low low normal tumor of the pituitary (adenoma), antibodies, environment, head injury,surgical removal6 adrenal glands(secondary) high2 low low low low3 normal low low normal tumor of the adrenal (adenoma), stress, antibodies, environment, Addison's disease, trauma, surgical removal (resection), miliary tuberculosis of the adrenal 1 Automatically includes diagnosis of secondary (hypopituitarism) 2 Only if CRH production in the hypothalamus is intact 3 Value doubles or more in stimulation 4 Value less than doubles in stimulation 5 Most common, does not include all possible causes 6 Usually because of very large tumor (macroadenoma) 7 Includes Addison's disease Treatment ;Adrenal crisis Intravenous fluids[4] Intravenous steroid (Solu-Cortef/injectable hydrocortisone) later hydrocortisone, prednisone or methylpredisolone tablets[4] Rest ;Cortisol deficiency (primary and secondary) Hydrocortisone (Cortef) Prednisone (Deltasone) Prednisolone (Delta-Cortef) Methylprednisolone (Medrol) Dexamethasone (Decadron) Hydrocortisone granules in casules for opening (Alkindi)[26] ;Mineralocorticoid deficiency (low aldosterone) Fludrocortisone acetate (To balance sodium, potassium and increase water retention)[4] See also Addison's disease - primary adrenocortical insufficiency Cushing's syndrome - overproduction of cortisol Insulin tolerance test - another test used to identify sub-types of adrenal insufficiency Adrenal fatigue (hypoadrenia) - a term used in alternative medicine to describe a believed exhaustion of the adrenal glands References ^ a b Eileen K. 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