***A REVIEW OF THE RADIOLOGICAL IMAGING OF ADRENAL LESIONS ENCOUNTERED IN CURRENT MEDICAL PRACTICE***

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ABSTRACT

*Modern radiological technology has transformed the way adrenal lesions are currently investigated. The contemporary radiologist has been catapulted to the forefront in the management of adrenal disease. With the increasing use of cross-sectional imaging, adrenal lesions are being serendipitously discovered in radiological studies undertaken for non-adrenal related conditions; the so-called “adrenal incidentaloma.” This review discusses the imaging modalities available for the characterization of these lesions, highlighting the current concepts and controversies in differentiating benign from malignant pathology. The article also provides a brief overview of the spectrum of adrenal pathology commonly encountered in the adult population.*

“I’m unaware that any modern authority has ventured to assign to them any

special function or influence….”

Dr Thomas Addison-1855

Tremendous strides have been made in the world of medicine since those words uttered by Dr Thomas Addison in 1855[1]. Despite their size and obscure structure, the adrenal glands are considered amongst the most vital organs in the human body. They are also an important site? for disease processes, harboring a myriad of pathologies.

Historically, adrenal lesions were discovered mainly at surgery or autopsy. However, with the advent of modern technology and the increasing use of advanced cross-sectional imaging techniques, the pendulum has swung. Adrenal lesions are now being detected incidentally in radiological examinations performed for other abdominal, non-adrenal related conditions.[2] This poses a diagnostic and clinical dilemma to the radiologist, clinician and patient alike. The medical fraternity seeks to characterize these lesions using non-invasive measures. Perhaps, the most challenging question is whether the adrenal “incidentaloma” is benign or malignant.

Adrenal masses are estimated to occur in approximately 9% of the general population. Approximately 5 to 8% of Computed Tomography(CT) and Magnetic Resonance Imaging (MRI) studies of the abdomen demonstrate incidentally detected adrenal lesions. The vast majority of adrenal incidentalomas are benign, non-hyperfunctioning and require no treatment.[2] Adrenocortical adenomas are the most commonly encountered adrenal incidentaloma, with a reported prevalence of 3-8.7%.[3]. of patients

Most incidental adrenal lesions in patients who do not have a background history of malignancy are benign. Indeed, in this subgroup of patients the incidence of adrenal malignancy approaches nil[4]. However, a clinical problem is encountered in patients with known extra-adrenal primary malignancy, raising the probability of metastatic disease. The clinical and radiological challenge is greatest in this subgroup of patients, as inaccurate characterization of the adrenal lesion has severe consequences. In these oncology patients with detected adrenal masses, approximately 50% have adrenal metastasis on biopsy[5]. In the setting of widespread metastatic disease, it is not critical if the adrenal gland is one of many sites of metastasis. However, if the adrenal gland is the solitary focus of spread, accurate characterization is imperative, as it advances the patient’s stage of disease, impacting on treatment and prognosis. Conversely, reporting a benign adrenal adenoma as metastases will imply advanced disease, potentially denying the patient definitive treatment and potential cure[1].

**IMAGING MODALITIES**

Imaging of adrenal pathology must be guided by the patient’s clinical and biochemical profile. The intracellular lipid content, perfusion dynamics and metabolic activity of an adrenal mass are key properties that underpin adrenal imaging.

**A. COMPUTED TOMOGRAPHY**

CT is the cornerstone of adrenal imaging. Morphology, CT densitometry, washout percentage and distant spread are crucial determinants that help characterize an adrenal mass and guide diagnosis.

MORPHOLOGICAL FEATURES

SIZE: Size of an adrenal incidentaloma is an important variable in assessing malignant potential. Larger lesions are more likely to be malignant. In an adrenal lesion >4cm, the chance of malignancy approaches 70%; and if >6cm approaches 85%. Traditionally, tumours >6cm were surgically resected owing to its high malignant potential. However, most centers of endocrine surgery now recommend 4cm as the threshold for adrenalectomy. Exceptions are myelolipomas which can present as large masses, but its signature CT appearance allows for confident diagnosis.[2]. Size, as a criterion alone is limited, as it does not distinguish malignant from benign lesions with 100% accuracy. It should therefore, always be reconciled with other radiological phenotyping as a predictor of malignancy.

STABILITY: Prior/serial imaging is of cardinal value in assessing the stability of a lesion. Stability/slow growth of a lesion over a 6-month period often signifies benignity. Conversely, a rapidly growing lesion is often malignant. A caveat is haemorrhage into a benign lesion which can result in abrupt change in size.[6]

INTERNAL CHARACTERISTICS: Morphological features are non-specific and considerable overlap between benign and malignant lesions exists. A large lesion with heterogeneous attenuation and irregular contours is suspicious of malignancy, as compared to a small, well-defined, smoothly marginated, homogenous lesion which favours benignity.[4] Large areas of intralesional necrosis are often associated with malignancy. Calcification and haemorrhage are non-specific features, seen in both benign and malignant lesions.

CT DENSITOMETRY

CT densitometry is the workhorse in the adrenal radiological assessment. Approximately 70% of adrenal adenomas are lipid-rich and contain abundant intracytoplasmic fat, as opposed to most malignant lesions that lack intracellular lipid. Lee et al were the first to report the importance of unenhanced CT densitometry in differentiating adenomas from non-adenomatous lesions.[7] The high lipid concentration lowers the density of most adenomas. Korobkin and colleagues reiterated this finding; demonstrating an inverse linear relationship between the lipid content of an adrenal lesion and the CT attenuation on unenhanced images. Conversely, almost all non-adenomatous lesions were deficient in intracellular fat and exhibited higher CT attenuation values.[8] Later, Boland in his meta-analysis concluded that a Hounsfield Unit(HU)<10 on unenhanced CT had a 71% sensitivity and 98% specificity in diagnosing adenomas.[9] To date, this threshold value of <10HU has strong support in clinical practice and is the most widely endorsed standard value. It is proposed that any adrenal lesion with a HU<10 is probably benign and no further investigations or serial follow-up is required. Furthermore, Blake and colleagues recognized that a non-contrast density of >43HU is highly suspicious of malignancy.[5]

*Technique*: The densitometry measurement must be made through the center of the lesion to prevent partial volume averaging of adjacent retroperitoneal fat. A circular region of interest(ROI) should cover at least 1/2 to 2/3 of the surface area of the lesion.

*Limitations:*

\*Approximately 30% of adenomas are lipid-poor;lipid sensitive imaging techniques may therefore be less accurate. These lesions will often display unenhanced HU values >10.

\*A small percent of malignant lesions e.g. adrenal carcinomas, metastases and phaeochromocytomas may contain fat and have a HU<10.

\*Many adrenal lesions are detected incidentally on studies performed with contrast only; hence no images are available for unenhanced densitometry measurement.[2]

CT WASHOUT

Other CT parameters used to distinguish adenomatous from non-adenomatous lesions exploit the different perfusion dynamics and washout characteristics of adrenal lesions. Contrast in a benign lesion tends to wash out rapidly, whilst malignant lesions retain contrast for longer periods. It is proposed that this occurs because of increased microvascularity and capillary permeability, resulting in leakage of contrast into the extravascular space of malignant lesions. There are 2 percentage washout methods employed in clinical practice:

*\*If an* ***unenhanced scan*** *was obtained, then an* ***absolute percentage washout*** *value (APW) is calculated.*

*\*If* ***no unenhanced*** *scan is available, a* ***relative percentage washout*** *value (RPW) is calculated.*

(Table1)

Show how these are calculated-(This is shown in the example of an incidentaloma in Fig 14a-c.

The washout protocol uses the post-contrast CT attenuation values obtained at 60 seconds after intravenous contrast administration and a delay of 15minutes. An APW>60% or RPW>40% is compatible with a benign adrenocortical adenoma(Fig1a-c). Conversely, a lesion with an APW<60% or RPW<40% is almost always malignant.[10]Contrast washout is independent of the lipid content of an adrenal lesion, making it one of the key standard imaging investigations. This superior technique has found prime position in imaging the category of lipid-poor adenomas.[6]

10 MIN CT WASHOUT

To aid scanning workflow, some investigators have proposed the delayed contrast scan in the washout protocol be performed at 10 minutes. However, Sangawaiya et al recently revisited the accuracy of the 10min delay scan and reported suboptimal sensitivities in its detection of adenomas. It is therefore recommended that the 15min delay continues to be endorsed in standard clinical practice.[11]

LOCO-REGIONAL AND DISTANT SPREAD

Vascular extension, invasion of surrounding structures, loco-regional lymphadenopathy and metastases are findings compatible with malignancy.

CT HISTOGRAM AND DUAL ENERGY CT:

Most studies have shown these two CT techniques to have low sensitivities, limiting their use in routine clinical practice. These methods are reserved as adjunct tools in evaluation of the “indeterminate” adrenal lesion.[5]

*CT Histogram* - Like unenhanced CT densitometry, CT histogram analysis is based on the intracytoplasmic lipid content in an adenoma. Whilst conventional CT densitometry is a measure of the mean attenuation of a lesion, a CT histogram has the added advantage of assessing the distribution of tissue attenuation within a mass. CT histogram analysis entails placing a ROI within the adrenal lesion and then post-processing each pixel with a histogram analysis tool present on most viewing workstations. The CT histogram generated is a graphic display of the pixel attenuation values within a prescribed ROI plotted along the X axis against the frequency/number of each pixel along the Y axis. The percent of negative pixels of fat attenuation(values measuring < 0 HU) is then calculated. Bae et al proposed that a threshold value of more than 10% negative pixels is highly specific for adenomas[5].

*Dual Energy CT (DECT)* – This CT technique allows for images to be acquired with two different energies of 80Kv and 140Kv respectively. Lipid-containing lesions demonstrate a decrease in attenuation as the tube voltage setting decreases. Gupta et al recognized that a decrease in attenuation of an adrenal mass between 140Kv and 80Kv is highly specific of an adrenal adenoma. In comparison, adrenal metastases demonstrated an increase in attenuation on DECT [5,10].

Furthermore, new DECT scanners are able to reconstruct *virtual* unenhanced images from contrast enhanced CT data, by subtracting the iodine content. In daily practice, many abdominal scans are performed after the administration of intravenous contrast. Furthermore, a 15 minute delay scan is at times difficult to obtain as patients have often left the CT department, prior to the scan being reviewed. Characterization of an adrenal incidentaloma in these cases is not possible. Virtual reconstructed unenhanced images thus allow for unenhanced CT densitometry to be performed for lesion characterization without the need for the patient returning for a repeat scan and obviates a repeat radiation dose[10].

[Explain a bit more what these entail]

**B. MAGNETIC RESONANCE IMAGING**

CHEMICAL SHIFT IMAGING

Chemical shift imaging (CSI) is the principle technique employed in MR evaluation of adrenal lesions. It uses out-of-phase (OP) and in-phase (IP) techniques. Similar to CT densitometry, CSI exploits the presence of abundant intracellular lipid in adenomas that helps distinguish them from non-adenomatous lesions. The basis of CSI is the existence of different resonant frequencies of hydrogen protons of water and fat within a given voxel. Thus, on out-of-phase imaging, the net effect is a cancellation of signal between lipid and water protons within a voxel. Therefore, lesions such as adenomas that contain almost equal voxel concentrations of lipid and water exhibit complete signal intensity loss/drop-off on OP and appear darker when compared to the IP image(Fig2).[6]

To qualitatively evaluate chemical shift change and assess signal drop-off visually, comparison to an internal standard reference is helpful. In clinical practice, the spleen is used as the internal reference organ. The liver should not be used as reference as many patients have incidental diffuse fatty hepatic infiltration which will also exhibit signal drop on OP, resulting in erroneous results. The sensitivity and specificity of CSI for distinguishing benign from malignant lesions are reported at 78-100% and 87-100% respectively.[5]

The CSI signal loss can be calculated quantitatively by measuring the adrenal to spleen chemical shift ratio (ASR) or the adrenal signal intensity index (ASII). Measurements of <0.71 on ASR or >16.5% on ASII are consistent with an adenoma.[10]

(Table2)

Most studies have shown no significant difference between CT densitometry and CSI in characterizing lipid rich adenomas. However, CSI is superior in evaluating lipid-poor adenomas that measure between 10-30 HU on unenhanced CT.[5]

*Limitations:* Adrenal carcinomas, phaeochromocytomas and clear cell renal cell carcinoma metastases can contain variable amounts of fat and may demonstrate signal loss on OP images.

DIFFUSION WEIGHTED IMAGING (DWI)

In theory, malignant lesions should demonstrate lower apparent diffusion coefficient (ADC) values and restricted diffusion. Despite resounding success in the evaluation of tumours elsewhere in the body, DWI has not shown promising use in differentiating malignant from benign adrenal lesions, as evidenced by the recent study by Sandrasegren et al.[12]

MR SPECTROSCOPY

Based on specific pattern change in metabolite concentration, spectroscopy has shown some promise in adrenal lesion characterization. Although not in widespread clinical use, threshold values of 1.2 for choline-creatine ratio, 0.38 for choline-lipid ratio and 2.1 for lipid-creatine ratio enabled distinction of adenomas and phaeochromocytomas from adrenal carcinomas and metastases.[6]

[What about a fat-sat sequence?] I did not include this sequence as there is little clinical use for fat sat as adenomas have intracytoplasmic lipid and not macroscopic fat. Its macrosopic fat that will suppress.

**C. POSITRON–EMISSION TOMOGRAPHY(PET)**

PET plays an increasingly pivotal role in functional imaging, governed by the metabolic activity of the adrenal lesion. Like most other non-benign lesions, malignant adrenal neoplasms show increased 18F-fluoro-deoxyglucose(18FDG) activity due to increased glucose utilization. The strength of PET lies in its accurate ability to assess physiological change, which often precedes gross anatomical changes that are detected much later.

Excellent results observed by Boland in a meta-analysis, reported a 97% sensitivity and 91% specificity in distinguishing benign from malignant lesions.[13] The use of hybrid PET-CT has improved the diagnostic yield through including CT densitometry, morphological features and accurate localization. Qualitative PET analysis using visual comparison to liver uptake is used more commonly. Quantitative SUV analysis is of limited diagnostic use.

PET-CT, although a supreme diagnostic tool, is not without limitations:

\*It is less sensitive in detecting and characterizing small lesions, particularly those less than 1cm. \* A small percent of adenomas and infective lesions are mildly FDG avid.

\*False negatives may be encountered in adrenal metastases from primary malignancies that are non-FDG avid e.g. bronchoalveolar carcinoma, carcinoid tumours.

Other primary agents(F-fluoro-dopamine, 11C-hdroxyephedrine, F-DOPA,) are also in use, particularly for the diagnosis of phaeochromocytomas.

**D. ADRENAL SCINTIGRAPHY**

Scintigraphy provides functional characterization of the adrenal gland based on uptake and accumulation of radiotracer. Adrenomedullary agents eg. metaiodobenzylguanidine(MIBG) and adrenocortical agents eg.NP59(iodomethylnorcholesterol) are the two major categories of radiopharmaceuticals in use. MIBG is a structural and functional analogue of norephedrine, taken up by adrenergic neoplasms including phaeochromocytomas, neuroblastomas and paragangliomas [what about neuroblastomas?]. Whole-body imaging allows for the detection of multifocal disease, extra-adrenal phaeochromocytomas(paragangliomas), metastatic disease and residual/recurrent tumour. Octreotide, a somatostatin analogue is occasionally used for the evaluation of medullary disorders, but carries a lower sensitivity of approximately 30% in the detection of phaeochromocytomas.[6] N59 is the main radio-isotope employed in adrenal cortical scintigraphy. It is a cholesterol analogue that binds to lipoprotein receptors of adrenal cortical cells. Adenomas, with intact steroidgenesis show uptake of NP-59 whereas malignant and non-adenomatous lesions do not.

**E. ULTRASONOGRAPHY**

Owing to its widespread use, many adrenal masses are discovered incidentally on abdominal ultrasound. Although ultrasound has a limited role in adrenal lesion characterization, it is reliable in detecting size, assessing serial growth and determining the solid or cystic nature of an adrenal mass (Fig3).

**F. PERCUTANEOUS ADRENAL BIOPSY**

Radiological advances in dedicated adrenal imaging has allowed for more accurate non-invasive characterization of adrenal neoplasms, thereby reducing the number of adrenal biopsies performed. To establish a definitive diagnosis, biopsies are still performed for adrenal lesions that remain indeterminate on imaging. Percutaneous biopsies carry a complication rate of 8-12.7%. Complications include bleeding, pancreatitis, pneumothorax, infection and needle tract seeding. Biopsy of an unsuspected phaeochromocytoma carries the potential risk of precipitating a cathecolamine storm. Biochemical testing to exclude a possible phaeochromocytoma is therefore advocated prior to undertaking any adrenal biopsy.[2]

**G. VENOUS SAMPLING**

Selective adrenal vein sampling is performed infrequently. It is invasive and requires specialized expertise. Venous sampling is used to localize the source of adrenal hormonal secretion, especially in the evaluation of hyperaldosteronism.

**ADRENAL PATHOLOGY**

When classifying an adrenal neoplasm, it is imperative to broadly establish if the lesion is hyperfunctioning or non-hyperfunctioning, and whether it is benign or malignant.

Hyperfunctioning lesions, although rare, can be potentially fatal if unrecognized. Whilst these lesions usually present with a characteristic clinical syndrome, the disease may remain occult and subclinical. Conditions with excessive hormone production include:

\*Cushing Syndrome: increased cortisol secretion

\*Conn Syndrome: increased aldosterone production

\*Adrenogenital Syndrome: androgen overproduction

\*Phaeochromocytoma: excess catecholamine secretion

**ADRENOCORTICAL ADENOMAS**

Adenomas are the most frequently encountered adrenal cortical lesion, representing 80% of all adrenal neoplasms. The prevalence of adenomas increases with age, occurring in approximately 0.2% of CT scans in patients aged 20-29yrs; escalating to 7-10% in the elderly.[2]

Adenomas are benign and usually non-hyperfunctioning. A small percent of adenomas are however active, resulting in a hyperfunctioning syndrome. 10-20% of adenomas are bilateral.[14] Adenomas measure between 1-3cm in size. Most are round/oval in shape, well-defined, smooth margined, homogenous and stable/slow growing. Calcification, haemorrhage and necrosis are rare but may be present in larger lesions. Atypical adenomas can appear heterogeneous, irregular and large.

70% of adenomas are lipid rich with abundant intracellular fat. These lesions display classic imaging characteristics(refer to Table 3).The 30% of adenomas that are lipid poor will have HU >10 and may not conform to the classic imaging findings. CT washout, chemical shift imaging and PET will further help differentiate this subgroup of adenomas from non-adenomatous lesions.

**METASTASIS**

Metastasis is the most common malignant lesion affecting the adrenal gland. At autopsy, it is found in approximately 27% of cancer patients.[3] The adrenal gland is the 4th most common site for overall metastatic disease. Primary tumours that commonly metastasize to the adrenal gland include bronchogenic, breast, thyroid andcolon carcinomas and melanoma.[14] 50% of adrenal metastases are bilateral. When small, adrenal metastases have no specific features. However, larger lesions are ill-defined with irregular margins and display heterogeneity owing to areas of haemorrhage, necrosis and calcification.

**ADRENOCORTICAL CARCINOMA**

Primary adrenal carcinoma (ACC) is a rare, aggressive malignancy arising from the adrenal cortex. It has a bimodal age distribution; affecting children <5yrs of age and adults in their 4th -5th decade.[10]. Most ACC are hormonally active. ACC is associated with various syndromes including Carney complex, Beckwith-Wiedemann Syndrome, MEN-I and Li-Fraumeni Syndrome.[10] These tumours are large, often exceeding 6cm.[2] Most ACC demonstrate aggressive features with vascular invasion, local infiltration, distant metastases and retroperitoneal lymphadenopathy.[14] ACC have irregular margins and can display necrosis, intratumoral haemorrhage and calcification. These tumours enhance avidly and characteristically have a rim of peripheral nodular enhancement. ACC retain contrast resulting in APW<60% and RPW<40%. MRI demonstrates variable heterogeneous T1and T2 signal intensities and shows no significant chemical shift change. MRI better evaluates tumour extension into the IVC and renal veins. ACC is FDG avid(Fig5a,b). PET has the added advantage of detecting metastatic spread.

**PHAEOCHROMOCYTOMA**

Phaeochromocytomas are neuroendocrine catecholamine-secreting tumours that arise from chromaffin cells of the adrenal medulla or sympathetic paraganglia. Phaeochromocytomas are dubbed the “10% tumour” as 10% are extra-adrenal, 10% bilateral, 10% malignant and 10% familial(Fig6). Most phaeochromocytomas are sporadic but there is a strong association with various syndromes(Von Hippel-Lindau, Neurofibromatosis1, MEN II)[10] Clinically, patients present with paroxysmal hypertension, palpitations, flushing, diaphoresis and elevated levels of cathecolamines, vanillylmandelic acid and metanephrines.

Phaeochromocytomas have variable imaging characteristics, with a reported size of 1.2-15cm (mean size 5.5cm). Smaller lesions are homogenous and display a density of 40-50HU.[14] Larger lesions have a heterogeneous appearance with areas of cystic necrosis, myxoid degeneration, haemorrhage and calcification(Fig7a). Phaeochromocytomas enhance avidly. Their washout dynamics are variable and inconsistent. Most phaeochromocytomas (irrespective of their malignant potential) demonstrate APW<60% and RPW<40%. However,some phaeochromocytomas display CT washout values that mimic adenomas.

Historically, the use of intravenous ionic contrast in patients with phaeohromocytomas was guarded; as it could potentially precipitate an adrenal crisis. However, recent experience shows no adverse events with non-ionic contrast, eliminating the need for premedication.[10]

MIBG has a high sensitivity and specificity in detecting phaeochromocytomas (Fig7b). The advantage of scintigraphy is that extra-adrenal phaeochromocytomas and metastases can be detected. Increased FDG activity is noted in phaeochromocytomas. Recent studies also suggest a role for PET in detecting MIBG-negative phaeochromocytomas.[6]

Phaeochromocytomas show intermediate to high T2-weighted signal intensity (Fig8a,b). The classic “light bulb” T2 hyperintensity that was thought to be a characteristic feature, is only present in 34% of phaeochromocytomas.[14] Imaging plays a vital role in demonstrating local invasion and metastatic spread , the only reliable criteria that help establish a diagnosis of malignant phaeochromocytoma(Fig9a-c).

[T2 hyperintensity is present even in the absence of necrosis]

**ADRENAL LYMPHOMA**

Primary adrenal lymphoma is rare with less than 100 reported cases. Secondary lymphomatous involvement of the adrenal gland occurs typically with non-Hodgkin’s lymphoma, and is seen in 4% of CT studies with disseminated disease.[14] 43% of adrenal lymphoma is bilateral.10] The CT appearance ranges from a discrete mass to diffuse infiltration, whereby the adrenal gland is characteristically enlarged but maintains its adreniform shape(Fig10). Calcification is rare, occurring only post therapy. MRI characteristics are variable but parallel the imaging features of metastases. Adrenal lymphoma demonstrates marked increased FDG activity.

**MYELOLIPOMA**

Myelolipoma is a benign, non-functioning adrenal neoplasm composed of an admixture of mature adipose tissue and haemopoeitic elements. Myelolipomas are usually unilateral and vary in size, some of which grow extremely large.

Macroscopic fat is the hallmark feature. CT demonstrates a well circumscribed, heterogeneous mass with areas of fat density and scattered amounts of soft tissue myeloid elements (Fig11a). A pseudocapsule compromised of a thin rim of compressed adrenal cortex is seen in most myelolipomas. Calcification is noted in approximately 24% of lesions.[10] On ultrasound, myelolipomas display doppler flow and heterogeneous echogenicity with the fatty components being characteristically echogenic.(Fig11b).

On MRI , the diagnosis can be confirmed with the fatty elements demonstrating T1 hyperintensity and suppression following fat saturation. An “India-ink” artifact may be seen as a sharp black line outlining the fat-adrenal interface on chemical shift imaging.(Fig11c). The presence of macroscopic fat is not solely exclusive to myelolipomas, as very rarely other adrenal lesions(ACC, phaeochromocytomas and metastatic clear cell renal carcinomas) may contain fat.

**INFECTION**

Granulomatous infections affecting the adrenal gland are often secondary to tuberculosis or histoplasmosis. Involvement is usually bilateral but asymmetric. It is frequently associated with adrenal insufficiency. Imaging characteristics are non-specific and include adrenal gland enlargement, soft tissue masses, cystic change, heterogeneous enhancement and calcification(Fig12).[10]

**ADRENAL HAEMORRHAGE**

Adrenal haemorrhage may result from trauma, coagulopathies, venous thrombosis, in neonates, orthotopic liver transplantation and stress related to surgery, sepsis or hypotension. 20% of haematomas are bilateral.[10] Acute haematomas appear as oval/round high attenuation(50-90HU)masses.[2] On serial imaging, these lesions decrease in size and attenuation. MRI appearance varies with the age of the haematoma. Areas of T1 hyperintensity are present in acute haemorrhage with a characteristic haemosiderin hypointense rim noted in the subacute stage. Chronic haematomas are T1-weighted and T2-weighted hypointense. Gradient echo sequence is sensitive to the detection of blood products. The magnetic susceptibility results in a pronounced T2 signal loss that visually accentuates the haemorrhagic focus; the so-called “blooming artifact”. -[explain blooming a bit]

Also liver transplants and neonates?

**ADRENAL CYSTS**

Three types of adrenal cysts are identified:

\*Endothelial cysts are the most common and are simple in nature. They have thin walls, a HU<20, no enhancement and are T2 hyperintense.

\*Pseudocysts- are usually secondary to a previous insult eg. haemorrhage, infarction. These cysts are complex with thick walls, internal septations, solid components, curvilinear calcification and haemorrhagic products.

\*Parasitic cysts are often secondary to ecchinococcal infection. Depending on the stage of disease the cyst can range from simple to a multilocular complex cystic lesion. Complex cysts can be difficult to differentiate from more sinister lesions such as adrenal abscess, cystic metastasis or necrotic adrenal neoplasms.[3]

**DIAGNOSTIC ALGORITHM**

The recent White Paper of the American College of Radiology Committee on Incidental Findings recommended a comprehensive approach to the management of an adrenal incidentaloma(Fig13).[15] Does such a guideline have place in the African health care context? In an already overburdened health care system, with limited resources, poor patient referral patterns and financial constraints, is it pragmatic to adopt first world recommendations? The answer is yes, as most adrenal lesions are successfully detected and accurately characterized on a single patient visit, using CT methods only. Very few lesions will require further evaluation by specialized techniques or expertise. The greater challenge is to ensure all imagers have an astute understanding of the modern principles, imaging modalities and specialized techniques available for the evaluation and management of adrenal pathology.

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