Review Article

Adrenal imaging (Part 2): Medullary and secondary adrenal lesions

Ekta Dhamija, Ananya Panda, Chandan J. Das, A. K. Gupta

Department of Radiodiagnosis, All India Institute of Medical Sciences, Ansari Nagar, New Delhi, India

ABSTRACT

Adrenal malignancies can be either primary adrenal tumors or secondary metastases, with metastases representing the most common malignant adrenal lesion. While imaging cannot always clearly differentiate between various adrenal malignancies, presence of certain imaging features, in conjunction with appropriate clinical background and hormonal profile, can suggest the appropriate diagnosis. The second part of the article on adrenal imaging describes adrenal medullary tumors, secondary adrenal lesions, bilateral adrenal lesions, adrenal incidentalomas and provides an algorithmic approach to adrenal lesions based on current imaging recommendations.

Key words: Adrenal imaging, adrenal incidentalomas, adrenal lymphoma, adrenal medulla, adrenal metastases, bilateral adrenal masses, pheochromocytoma

Introduction

With the advances in imaging techniques and protocols, detection of adrenal lesions has significantly increased. Adrenal nodules are seen in 9% of human population and most of them are incidentally detected during abdominal imaging for other conditions and are termed incidentalomas.[1] Most of these incidentally detected lesions are benign especially if the patient does not have any endocrine abnormality or malignancy.[2-4] Adrenal lesions can also be either primary or secondary and are classified in Table 1. Secondary adrenal lesions are lesions that do not arise directly from the adrenal cortex or medulla. The most common secondary adrenal lesions are metastases. Bilateral adrenal lesions are also secondary and are due to metastases, hemorrhage or granulomatous conditions. In the second part of the two-part review on adrenal imaging, adrenal medullary tumors, adrenal incidentalomas, secondary adrenal lesions and bilateral adrenal lesions are described followed by a summary of the current approach to adrenal lesions.

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Primary adrenal medullary tumors

Pheochromocytomas

Pheochromocytoma is paraganglioma of adrenal medulla, seen in 0.1-0.2% of patients presenting with hypertension. Patients may present with palpitations, headache, diaphoresis and flushing due to release of catecholamines by the tumor. It is also seen in association with various syndromes like multiple endocrine neoplasia (MEN) type 2, Von Hippel-Lindau (VHL), neurofibromatosis, tuberous sclerosis and Sturge Weber syndrome. Ten percent of these tumors are malignant or extra-adrenal in origin or non-functioning or bilateral (Rule of 10). Clinical diagnosis is done with increased levels of vanillyl-mandelic acid in a 24 hour urine sample or increased plasma catecholamine levels.[5-7] Malignant pheochromocytomas are diagnosed in the presence of metastases to non-chromaffin cell containing tissues such as bone, liver, lung and lymph nodes.[8,9]

Imaging of pheochromocytoma overlaps with adenomas and adrenocortical carcinoma (ACC) in many ways. The

Corresponding Author: Dr. Chandan J. Das, Department of Radiodiagnosis, All India Institute of Medical Sciences, Ansari Nagar, New Delhi - 110 029, India. E-mail: docchandan17@gmail.com

size and attenuation are variable (homogeneous if small and heterogeneous if large) and may contain fat or necrosis within. On contrast administration, the mass enhances avidly as compared to adenomas [Figure 1] with rapid washout (APW and RPW > 60% and 40%, respectively). MRI does not provide any additional information as there is considerable overlap between MR appearances of pheochromocytomas and other adrenal tumors. The so-called "light bulb" sign or T2 hyperintense signal of pheochromocytoma is less common than previously described[10] [Figure 2].

Malignant pheochromocytomas can contain cystic necrosis and hemorrhage but are usually smaller in size than ACC. Calcification is seen in 10% and inferior vena cava invasion is very rare in contradistinction to ACC. Thus, the presence of calcifications and/or venous invasion in a large adrenal mass should favor a diagnosis of ACC rather than a pheochromocytoma.[1]

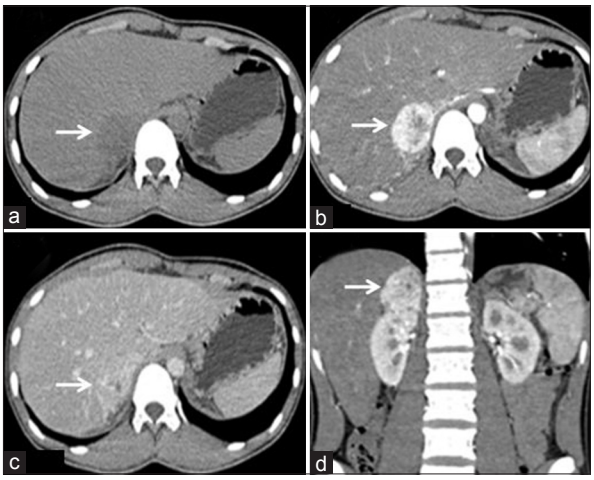

Figure 1: Washout in pheochromocytoma. NCCT (a), 1min post contrast (b) delayed- 15 minutes post contrast (c) axial sections and coronal reconstructed section (d) in a 19-years-male with right pheochromocytoma show well defined iso to hypodense lesion in right adrenal measuring 4.3 cms in size and 23 HU (not shown in image) on unenhanced scan with avid contrast enhancement (251 HU, not shown) with rapid washout (31HU)

Functional imaging plays an important role in imaging of a pheochromocytoma. MIBG (metaiodobenzylguanidine) is a structural analog of norepinephrine, a product released by pheochromocytoma. Hence, radionuclide imaging 24-72 hours after administration of MIBG show uptake [Figure 3] in the pheochromocytoma.[11] MIBG scan can also detect other sites of metastases in malignant pheochromocytomas. In non-MIBG avid lesions, FDG-PET or In-111-octreotide scans can show uptake in primary tumor and metastatic sites.[11,12]

Neuroblastomas/ganglioneuroblastomas/ganglioneuromas

These are neurogenic tumors arising from adrenal medulla. Neuroblastomas and ganglioneuroblastomas are seen in children while ganglioneuromas are seen in teenagers and adults. Neuroblastomas are malignant tumors, ganglioneuroblastomas are potentially malignant and ganglioneuromas are benign lesions with peak at 10 years of life. Neuroblastomas can secrete catecholamines, thereby presenting with diarrhea and hypertension. Neuroblastomas

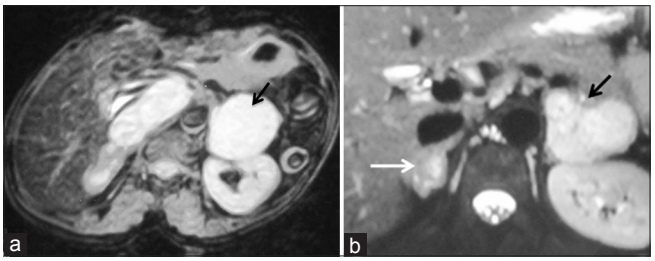

Figure 2: MRI of Pheochromocytoma. T2 weighted axial image (a) showing left suprarenal mass (arrow) which has uniform hyperintense signal intensity (light bulb sign). Similarly, bilateral hyperintense adrenal masses are seen in another patient with bilateral pheochromocytoma (b)

Table 1: Comprehensive classification of adrenal lesions based on etiology, cell of origin and hormone-secreting status

Adrenal lesions	Histologic origin	Benign	Malignant	Hormone excess	Syndrome
Primary	Cortical	Adenoma	Adrenocortical Carcinoma	Aldosterone (Zona glomerulosa) Glucocorticoids (Zona fasciculata) Sex horomones (Zona reticulata)	Conn's syndrome Cushing's syndrome Virilising syndromes
		Adrenal hyperplasia		Aldosterone (Zona glomerulosa) Sex horomones (Zona reticulata)	Conn's syndrome Virilising syndromes
		Oncocytoma	Malignant oncocytoma	Non-functioning	None
	Medullary	Pheochromocytoma	Malignant pheochromocytoma	Catecholamines	Symptoms of catecholamine excess such as palpitations, tachycardia, hypertension,
		Ganglioneuroma	Neuroblastoma Ganglioneuroblastoma		
Secondary	No specific histologic origin	Myelolipoma Cysts Lipoma Hemorrhage	Metastases Lymphoma	None	None
Other entities	Any cell origin/ Either primary or secondary	Incidentalomas Collision tumors (adenoma+myelolipoma)	Collision tumors (adenoma+ metastases)	None	None

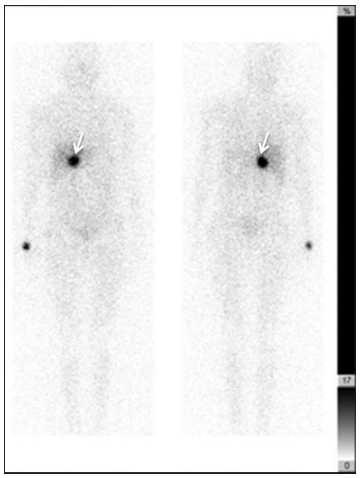

Figure 3: Functional imaging of pheochromocytoma. MIBG (Meta-iodo benzyl guanidine) scan shows uptake in right suprarenal location in a clinically and biochemically proven case of pheochromocytoma. Uptake in the right hand is the site of injection of radionuclide

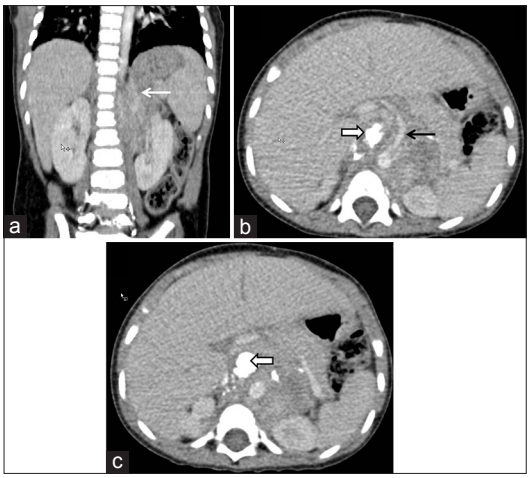

Figure 4: Neuroblastoma. Coronal reformatted (a) and axial CT images (b,c) of a one-year old girl show left suprarenal mass (arrow, a). The mass has coarse calcifications (block arrows), is crossing the midline and is encasing the aorta and celiac artery (arrow, b) consistent with neuroblastoma

can also present with various paraneoplastic syndromes. However, abdominal lump is the most common presentation of these tumors. On imaging, they present as suprarenal masses displacing kidney inferiorly. They also tend to encase the major blood vessels, cross the midline and extend into spinal canal. Calcification is seen in 80-90% of these tumors, being coarse or amorphous type in neuroblastomas and stippled or punctate type in ganglioneuroblastomas/ganglioneuromas [Figure 4]. Neuroblastomas frequently have areas of necrosis or hemorrhage while ganglioneuroblastomas/ganglioneuromas tend to be more homogeneous.[1]

Metastases in neuroblastomas commonly occur in lymph nodes, liver, bones and lungs. Neuroblastomas in infants can present with metastases exclusively to skin and bone marrow. Multiple modalities are used in tumor staging and include cross-sectional imaging, nuclear medicine MIBG and Tc-99 medroxyphosphonate (MDP) bone scans, FDG-PET and bone marrow sampling.[13,14] The role of CT is to chiefly delineate the tumor extent and staging.

MRI is useful in delineating intraspinal extension and for follow-up as it does not involve any radiation exposure.[14]

Adrenal incidentalomas

Adrenal incidentalomas are adrenal lesions greater than 1 cm in size detected incidentally on imaging, most commonly on computed tomography and occasionally on ultrasound, magnetic resonance imaging (MRI) or PET-CT.[15] Currently adrenal incidentalomas are considered to be the most common adrenal lesion; increased detection has been attributed to the widespread availability of CT and improvements in CT technology enabling detection of even small adrenal lesions.[15,16]

While the incidence of incidentalomas increases with age, it can be seen across all age groups. In the absence of known malignancy, an incidentaloma is nearly always benign with non-functioning adenomas being the most common incidentaloma.[15] Other benign masses like cysts, myelolipoma and hemorrhage also have characteristic imaging features and can be differentiated from the rest of the spectrum. However, in a patient with a known underlying malignancy, the probability of incidentaloma being malignant substantially increases and 50-70% of adrenal masses are likely to be metastatic.[17]

Alexandraki *et al*.[2,8] proposed a "Rule of Four" for adrenal incidentalomas, namely incidentalomas are detected in 4% of CTs across all age groups, 4% are due to malignant etiology, a size greater than 4 cm initiates a need for removal and 4 years is at the recommended period of follow-up.

Thus, a radiologist plays a vital role in further characterization of incidentalomas. Using various imaging techniques such as unenhanced CT scans, CT washout values, chemical shift (CS)-MRI and occasionally functional imaging, adrenal lesions can be classified as benign or malignant in most cases. In persistently equivocal lesions, surgical resection or tissue sampling may need to be done for definitive diagnoses.

Secondary benign lesions

These form heterogeneous spectrum and are often detected as incidentalomas. Their imaging characteristics are enumerated as follows:

Myelolipoma

A myelolipoma is an uncommon benign lesion occurring with a prevalence of 0.08-0.2%.[18] It is composed of mature

fat interspersed with marrow-like hematopoietic tissue, is usually unilateral and asymptomatic.[19] The lesion may occur in isolation involving adrenal gland with or without hemorrhage within or it may be seen in association with other adrenal diseases. The exact etiology of this entity is not known but metaplasia of the reticuloendothelial cells in the capillaries of adrenal gland occurring due to necrosis, infection or stress has been postulated as the etiopathogenesis for the same.[19]

The size varies from 2 to 13 cm and attenuation depends on the amount of fat with the presence of calcification in 24% of lesions. On ultrasound (USG), myelolipoma is visualized as heterogeneously hyperechoic lesion owing to the presence of variable amount of fatty and myeloid component. CT remains the diagnostic modality of choice which depicts the presence of fat as low attenuation areas (<10HU) within the lesion which are interspersed within the high attenuation areas of hematopoietic tissue [Figure 5]. The areas of fat are hyperintense on T1- and T2-weighted images (WI) on MRI with loss of signal on fat-suppressed sequences [Figure 6]. The presence of hemorrhage causes alteration of signal intensity on MRI sequences.[18] Lipoma and angiomyolipoma are other fat containing lesions which are to be kept as differentials.

The benignity of the lesion does not warrant immediate intervention and the lesion needs to be followed up to rule out the occurrence of a collision tumor. Fundamental characteristics which indicate the need for surgical intervention are the symptoms at presentation, size of the tumoral mass (>5 cm), and increase in the volume of the tumor on two consecutive imaging studies.[20]

Adrenal cyst

Adrenal cysts are incidentally detected lesions with female predominance (3:1) which are usually asymptomatic. USG depicts a well-defined lesion with anechoic contents in uncomplicated cysts and CT demonstrates the exact nature and extent of the lesion.[21] The cysts are homogeneous, well-defined, thin-walled lesions having attenuation similar to water [Figure 7]. They may appear hyperdense in the case of hemorrhage or infection within and show no enhancement on post contrast scan.

Adrenal hemorrhage

Adrenal hemorrhage is usually encountered in patients with blunt abdominal trauma where it affects right adrenal more than the left.[21] Attenuation on CT depends on the stage of hemorrhage—higher attenuation in acute stage becoming isodense to hypodense as the clot liquefies and may calcify in chronic stage.[22]

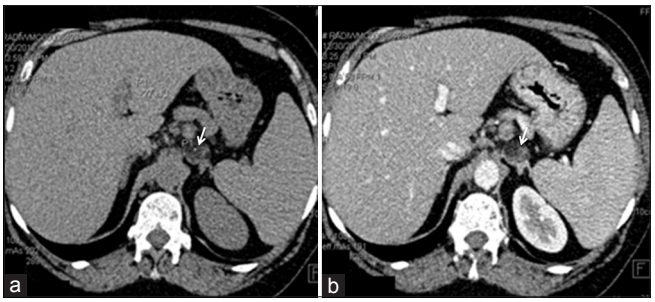

Figure 5: CT appearance of Myelolipoma. (a) Unenhanced CT axial section shows well definedhypodense lesion of left adrenal measuring 2.2 cms size, with mean HU of -27HU and no enhancement on post contrast scan (b) No further work up required for the patient pertaining to adrenal lesion as imaging suggests benign nature of the lesion (myelolipoma)

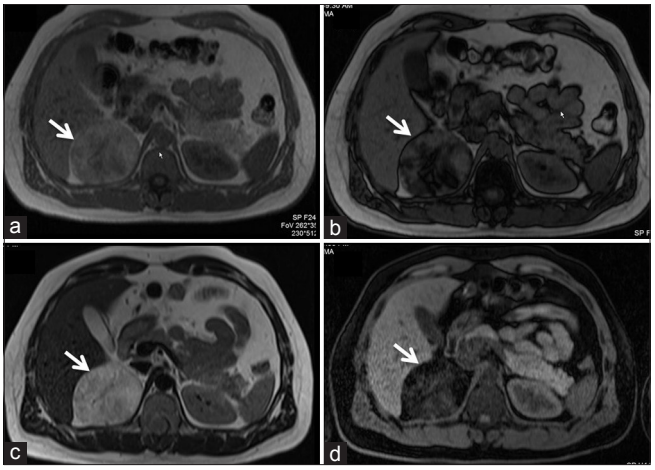

Figure 6: MRI appearance of Myelolipoma. T1 in-phase image (a) showing a large hyperintense right adrenal mass (arrow) with central hypointense areas. Out-of phase image (b) showing signal drop suggesting fat content in the mass. The mass appears hyperintense on axial T2 weighted image (c) Fat saturated T1 axial image (d) showing marked drop in signal. These features are suggestive of myelolipoma

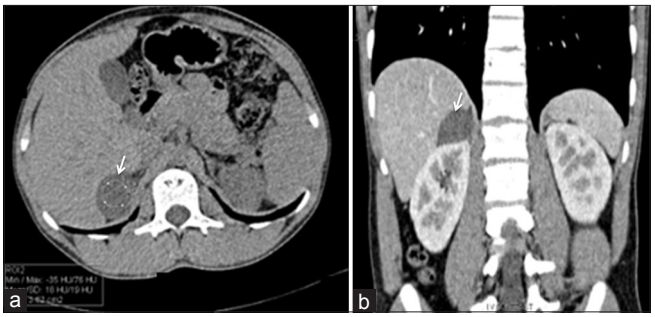

Figure 7: Right adrenal cyst. Unenhanced CT axial image (a) shows well defined homogeneously hypodense lesion in right adrenal with mean HU of 16 HU and no enhancement on post contrast scan (b) The lesion is stable in size and imaging characteristics for last 2 years

Non-traumatic adrenal hemorrhage occurs rarely and is associated with stress, hemorrhagic diathesis, coagulopathy or anticoagulation therapy and in the case of underlying adrenal tumors. On unenhanced CT, hemorrhage demonstrates attenuation values of 50-90 HU in acute

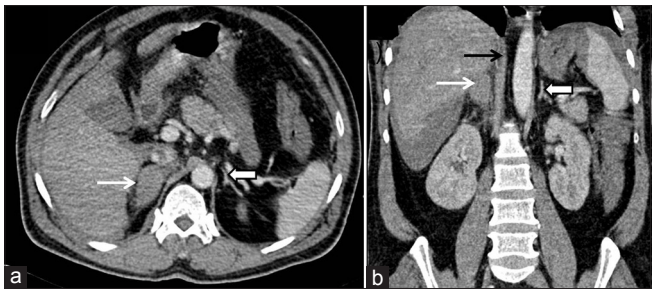

Figure 8: Adrenal hemorrhage. CECT axial (a) and coronal (b) images of a 34 year old man with trauma abdomen show a well-defined oval lesion replacing the right adrenal gland suggestive of adrenal hematoma (white arrow). There is also periadrenal fat stranding with mild thickening of right crus of diaphragm (black arrow, b). The left adrenal gland is normal in appearance (block arrows a,b)

and subacute stage with a gradual decrease in size and attenuation on follow-up imaging[22] [Figure 8].

MRI depicts the age of hemorrhage or hematoma which is isointense to slightly hypointense on T1 WI and hypointense on T2 WI in acute stage owing to the presence of intracellular deoxyhemoglobin and is hyperintense on both T1 and T2 WI by virtue of the presence of methemoglobin in the subacute stage. The presence of hemosiderin in chronic hemorrhage is seen as hypointense rim on T1 as well as T2 WI.[23]

Secondary malignant adrenal lesions

Metastases

The adrenal gland is the fourth most common organ to be involved by metastases after lung, liver and bone.[1] Metastases to adrenal glands have been variably described in gastrointestinal malignancies, lung, breast, prostate, kidney, liver tumors and melanoma, with carcinoma being the most common histological type.[6,17] Adrenal metastases are usually associated with multiple other organ metastases and isolated metastases to adrenal glands are uncommon.

Adrenal metastases on CT appear as soft tissue lesions either replacing the gland or as diffuse enlargement of the gland. Larger lesions are often heterogeneous and ill-defined [Figure 9]. Typically adrenal metastases show attenuation of more than 10 HU on non-contrast CT and APW and RPW of less than 60% and 40%, respectively. On chemical shift MRI (CS-MRI), metastases do not produce signal drop on opposed phase images. Using these criteria, metastases can be confidently differentiated from adenomas with a high degree of sensitivity and specificity.[1,11,24,25] However, pitfalls do exist that can obfuscate characterization of the adrenal mass. Differentiation of metastases from lipid-poor adenomas can be difficult as lipid poor adenomas also have attenuation

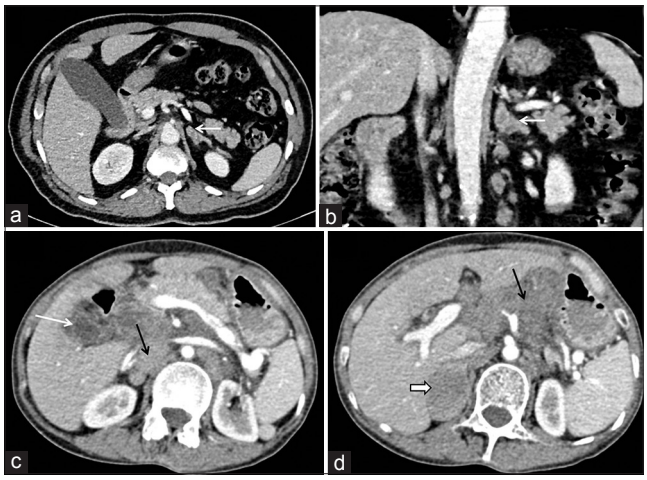

Figure 9: Adrenal metastases. (a,b): CT axial (a) and coronal reformatted image (b) of patient with primary lung cancer show bulky and ill-defined left adrenal gland (arrow) suggestive of metastases. (c,d): CT axial images of another patient with primary gall bladder cancer (white arrow, c) show right adrenal metastasis with mass replacing the adrenal gland (block arrow) and multiple metastatic retroperitoneal lymphadenopathy (black arrows, c,d)

more than 10 HU and do not demonstrate signal drop on CS-MRI. However, contrast CT can help in such cases as lipid poor adenomas also have APW and RPW of more than 60% and 40% similar to lipid-rich adenomas.[6] At times, metastases from hepatocellular carcinoma and renal cell carcinoma can also show signal drop on CS-MRI due to the presence of lipid rich cells. Also, liver and kidney tumors can also produce hypervascular adrenal metastases that may show APW and RPW of more than 60% and 40% similar to adenomas.[26] In such indeterminate lesions, FDG-PET CT can help in diagnosis as metastases are likely to be hyperrmetabolic as compared to adenomas. Also, if previous imaging is available, comparison of size from previous images and demonstration of increase in size definitively suggest metastases and obviate need for further testing.[25,27]

Collision tumor

Collision tumors refer to two histologically different lesions co-existing in the same gland without any significant tissue mixture. While both lesions in collision tumors can be benign (adenoma and myelolipoma, adenoma and lipoma), collisions of benign adenomas with malignant metastases or with ACC can occur[28] [Figure 10]. Adenoma–metastases collisions are more likely to be encountered due to the high incidence of adenomas and high propensity for adrenal metastases. These collision tumors can pose a dilemma even with all imaging techniques at the radiologist's arsenal as lesions show features of both adenomas (signal drop on CS-MRI and increased contrast washout) and metastases (absence of drop on CS-MRI, decreased contrast washout) on both CT and MRI. In such cases, FDG-PET/CT can help in

diagnosis by guiding targeted tissue sampling from the FDG avid part of the tumor.[1]

Lymphoma

Involvement of adrenal glands in lymphoma is uncommon and is usually secondary to non-Hodgkin's lymphoma (NHL) though primary adrenal lymphomas and secondary involvement in post-transplantation proliferative disorder have also been described.[29-31] Contrary to the high incidence of secondary adrenal metastases in carcinomas, secondary adrenal involvement in lymphoma is uncommon. The reported incidence of adrenal lymphoma varies from 1 to 4% in various described series, with nearly half of cases having bilateral involvement.[29,32]

On imaging, various manifestations of adrenal lymphoma have been described. In early stage, the adrenal glands may appear normal or mildly bulky on imaging that can be mistaken for normal appearance. Adrenal lymphoma can progressively appear as nodular hyperplasia, diffuse

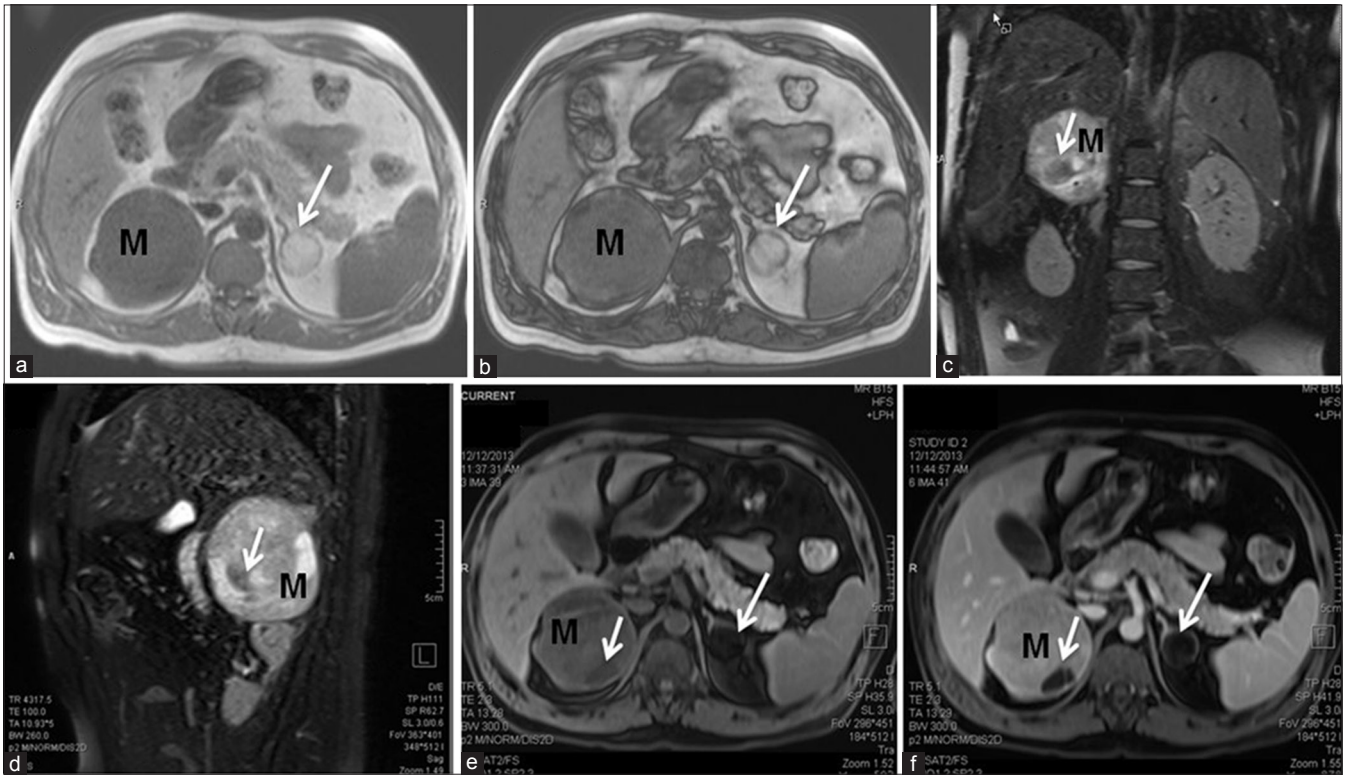

Figure 10: Collison tumor: Schwanomma with lipoma. T1 in-phase image (a) showing a large hypointense right adrenal mass (M) and hyperintense left adrenal mass (arrow). Out-of phase image (b) showing no signal drop of adrenal mass. Coronal (c) and sagittal (d) fat saturated T2 weighted image show large hyperintense right adrenal mass (M) with hypointense area (arrow) within likely to represent fat. Fat saturated axial image (e) showing large mass (M) in the right adrenal with area of fat (arrow) and another completely fatty mass in left adrenal (arrow). Note complete suppression of left adrenal lesion in image (c). Contrast enhanced image (f) shows enhancement of the right adrenal mass (M) with nonenhancing fatty area (arrow) and complete no enhancement of left adrenal mass (arrow). These features are suggestive of myelolipoma of right adrenal and lipoma of left adrenal. However, at surgery the right adrenal mass came out to be a schwanomma with separate adrenal lipoma suggesting a collision tumor

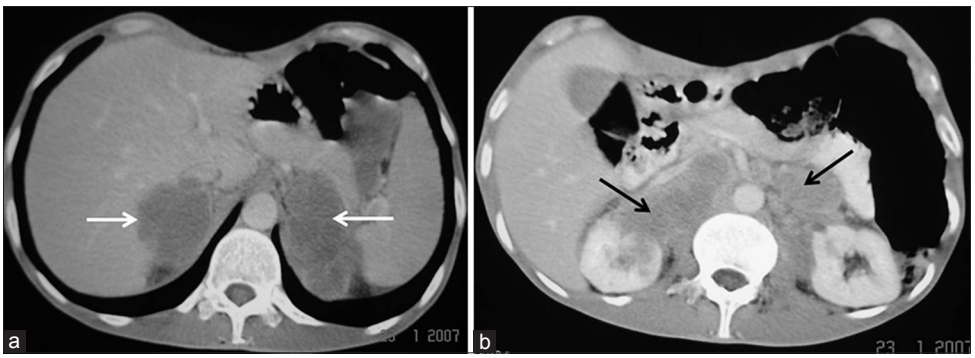

Figure 11: CT appearance of adrenal lymphoma CT axial images (a, b) in a patient with Non-Hodgkin's lymphoma show bilateral bulky, hypodense masses replacing the adrenal glands (arrows, a) and conglomerate retroperitoneal lymphadenopathy (arrows, b) consistent with adrenal lymphomas

infiltration of gland with maintained adreniform shape or as infiltrative soft tissue masses replacing the adrenal gland. At times the glands can be engulfed by bulky retroperitoneal lymphadenopathy and not seen separately [Figures 11 and 12]. When presenting as soft tissue masses, the CT and MR characteristics are non-specific with washout values, MR signal intensities and enlargement similar to that of adrenal metastases.[6,17,33] Diffusion-weighted imaging (DWI) is sensitive for adrenal lymphoma like lymphoma of other regions although DWI is not a standard imaging sequence for most of the adrenal lesions.[1] Diagnosis can be suspected in view of the clinical background of lymphoma and can be conclusively proved by tissue sampling. In contradistinction to metastases, adrenal lymphomas show good response to chemotherapy with decreased FDG uptake and even return to normal size after chemotherapy.[34]

Bilateral adrenal masses

Bilateral adrenal involvement has been described in both benign and malignant pathologies. The presence of bilateral adrenal masses narrows the differential diagnoses as bilateral adrenal lesions are more likely to represent metastases, lymphomas and granulomatous conditions like tuberculosis and fungal infections.[35] In the case of polytrauma and bleeding diathesis, patients can have bilateral adrenal hemorrhages. Neuroblastomas and pheochromocytomas can also be bilateral, with the later being bilateral in familial syndromes. Existence of adenoma on one side with ACC or myelolipoma on the other side have also been reported which are more coincidental in nature rather than representing any specific disease pathology.

The significance of bilateral adrenal masses lies in the high probability of an underlying malignant pathology as metastases can be bilateral in 49%[15,16] and lymphomas in 43%[28] and in their association with adrenal insufficiency and Addison's crises.[29,31] The presence of calcification in bilateral adrenal masses favor granulomatous conditions as calcification is uncommon in both metastases and lymphoma in the absence of prior therapy [Figures 13-15]. DWI may also be useful as malignant lesions can show restricted diffusion.[1] Finally, tissue sampling is useful as it can conclusively diagnose the pathology and can also guide specific anti-tubercular and anti-fungal therapy for granulomatous infections.

Conclusion

Adrenal glands can be affected by a wide spectrum of benign and malignant conditions. With the profusion

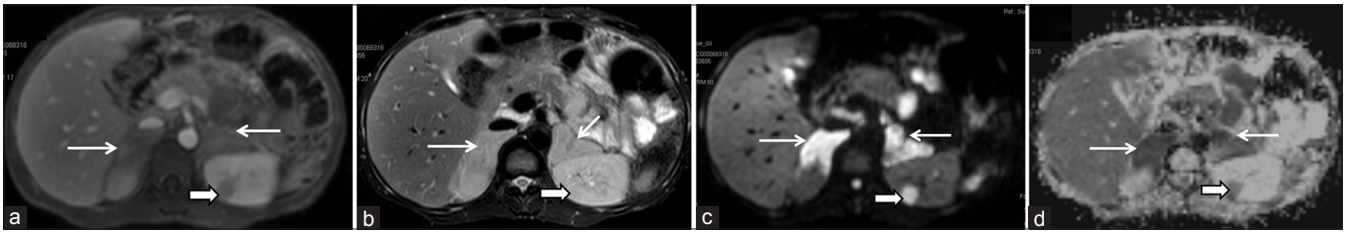

Figure 12: MR appearance of adrenal lymphoma. Axial T1 weighted (a), T2 weighted fat suppressed (b), diffusion weighted image (DWI) at b-value of 90 (c) and corresponding apparent diffusion coefficient (ADC) map (d) of patient with lymphoma show bilateral adrenal masses with maintained adreniform shapes (white arrows). The masses are hypointense on T1 (a), hyperintense on T2 (b) weighted images and show marked restriction of diffusion (hyperintense on DWI and dark on ADC) consistent with adrenal lymphomas. There is another focal lesion in left kidney (block arrow) which is hypointense on both T1 and T2 weighted images with restricted diffusion suggestive of renal lymphomatous deposit

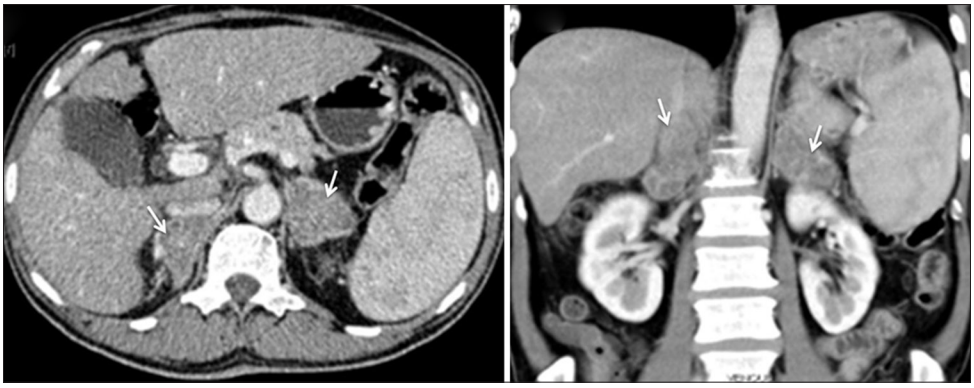

Figure 13: Bilateral adrenal masses-Tuberculosis. Bulky and lobulated contour of bilateral adrenals with maintained shape and outline showing hypodense areas within in a patient with abdominal discomfort. Diagnosis of tuberculosis was confirmed on histopathological examination

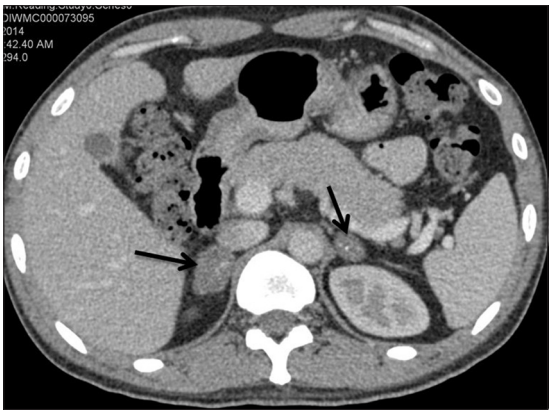

Figure 14: Bilateral adrenal masses-Histoplasmosis. Patient with chronic renal disease with bilateral well defined isodense large adrenal masses showing presence of soft calcification within. No significant enhancement seen on administration of contrast. Differential diagnosis includes granulomatous disorders, non functioning adenomas or metastases. Biopsy confirmed it to be histoplasmosis

of available imaging techniques, it has become possible to non-invasively differentiate benign and malignant adrenal lesions based on specific imaging features with a high degree of accuracy. Yet the appearances of various malignant adrenal lesions can be non-specific and overlap with each other. Size and heterogeneity remain the most predictive features of malignancy, as size more than 4 cm and presence of hemorrhage, necrosis, heterogeneous contrast enhancement and lack of contrast washout favor malignancy. However, in patients with a known extra-adrenal malignancy, the diagnostic algorithm is more geared toward proving the metastatic etiology of adrenal lesion irrespective of its size (<4 cm) either by imaging or direct tissue sampling. Similarly for bilateral adrenal masses irrespective of their size, tissue sampling and follow-up will be needed to prove the benign or malignant nature of the masses. The current recommendations for

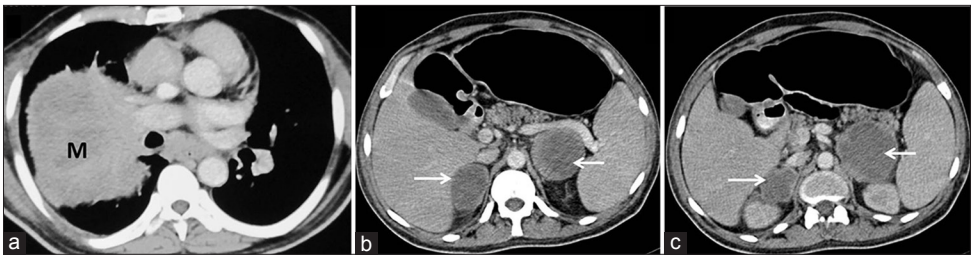

Figure 15: Bilateral adrenal masses- Metastases. CT axial images of chest (a) and upper abdomen (b,c) show mass in right lung (M) with tracheal and mediastinal vascular invasion and peripherally enhancing, predominantly necrotic bilateral adrenal metastases (b,c). The masses are displacing the inferior vena cava and splenic vein anteriorly on right and left side respectively consistent with displacement pattern produced by adrenal masses

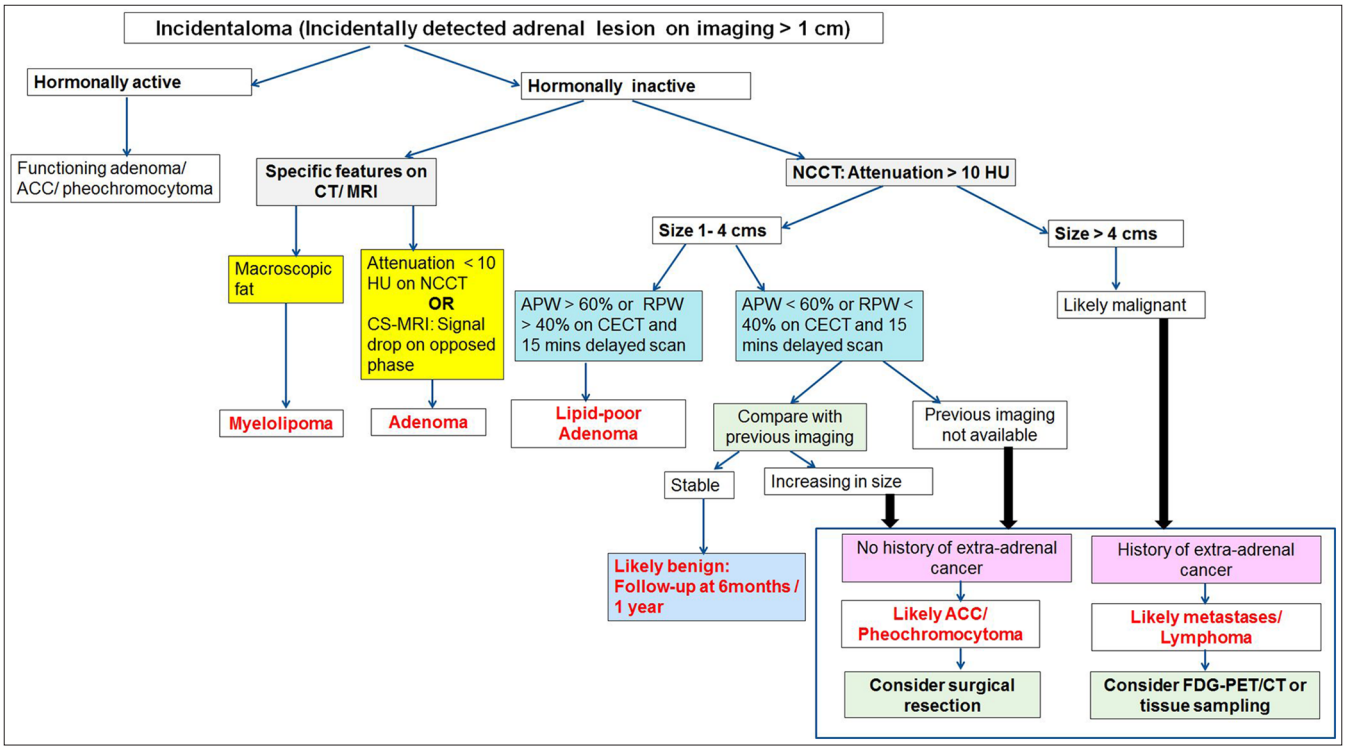

Figure 16: An algorithmic approach and current recommendations for imaging of adrenal masses

imaging incidental adrenal masses have been depicted in the flowchart [Figure 16].

References

1. Taffel M, Haji-Momenian S, Nikolaidis P, Miller FH. Adrenal imaging: A comprehensive review. Radiol Clin North Am 2012;50:219-43.
2. Das CJ, Baruah MP, Baruah UM. Radiological imaging in endocrine hypertension. Indian J Endocrinol Metab 2011;15 Suppl 4:S383-8.
3. Lee JE, Evans DB, Hickey RC, Sherman SI, Gagel RF, Abbruzzese MC, *et al*. Unknown primary cancer presenting as an adrenal mass: Frequency and implications for diagnostic evaluation of adrenal incidentalomas. Surgery 1998;124:1115-22.
4. Boland GW. Adrenal imaging: Why, when, what, and how? Part 3. The algorithmic approach to definitive characterization of the adrenal incidentaloma. Am J Roentgenol 2011;196:W109-11.
5. Krebs TL, Wagner BJ. MR imaging of the adrenal gland: Radiologic-pathologic correlation. Radiographics 1998;18:1425-40.
6. Johnson PT, Horton KM, Fishman EH. Adrenal mass imaging with multidetector CT: Pathologic conditions, pearls, and pitfalls. Radiographics 2009;29:1333-51.
7. Kawashima A, Sandler CM, Fishman EK, Charnsangavej C, Yasumori K, Honda H, *et al*. Spectrum of CT findings in nonmalignant disease of the adrenal gland. Radiographics 1998;18:393-412.
8. Eisenhofer G, Bornstein SR, Brouwers FM, Cheung NK, Dahia PL, Krijger RR, *et al*. Malignant pheochromocytoma: Current status and initiatives for future progress. Endocr Relat Cancer 2004;11:423-36.
9. Parenti G, Zampetti B, Rapizzi E, Ercolino T, Giachè V, Mannelli M. Updated and new perspectives on diagnosis, prognosis, and therapy of malignant pheochromocytoma/paraganglioma. J Oncol 2012;2012:e872713.
10. Jacques AE, Sahdev A, Sandrasagara M, Goldstein R, Berney D, Rockall AG, *et al*. Adrenal phaeochromocytoma: Correlation of MRI appearances with histology and function. Eur Radiol 2008;18:2885-92.
11. Mayo-Smith WW, Boland GW, Noto RB, Lee MJ. State-of-the-art adrenal imaging. RadioGraphics 2001;21:995-1012.
12. Chaudhary V, Bano S. Anatomical and functional imaging in endocrine hypertension. Indian J Endocrinol Metab 2012;16:713-21.
13. Balassy C, Navarro OM, Daneman A. Adrenal masses in children. Radiol Clin North Am 2011;49:711-27.
14. Rha SE, Byun JY, Jung SE, Chun HJ, Lee HG, Lee JM. Neurogenic tumors in the abdomen: Tumor types and imaging characteristics. Radiographics 2003;23:29-43.
15. Boland GW, Blake MA, Hahn PF, Mayo-Smith WW. Incidental adrenal lesions: Principles, techniques, and algorithms for imaging characterization. Radiology 2008;249:756-75.
16. Berland LL, Silverman SG, Gore RM, Mayo-Smith WW, Megibow AJ, Yee J, *et al*. Managing incidental findings on abdominal CT: White paper of the ACR incidental findings committee. J Am Coll Radiol 2010;7:754-73.
17. Lam KY, Lo CY. Metastatic tumours of the adrenal glands: A 30-year experience in a teaching hospital. Clin Endocrinol 2002;56:95-101.
18. Rao P, Kenney PJ, Wagner BJ, Davidson AJ. Imaging and pathologic features of myelolipoma. Radiographics 1997;17:1373-85.
19. Wani NA, Kosar T, Rawa IA, Qayum A. Giant adrenal myelolipoma: Incidentaloma with a rare incidental association. Urol Ann 2010;2:130-3.
20. Lesbats-Jacquot V, Cucchi JM, Amoretti N, Novellas S, Chevallier P, Bruneton JN. Lipomatous tumors of the adrenals: A report on 18 cases and review of the literature. Clin Imaging 2007;31:335-9.
21. Dunnick NR, Korobkin M. Imaging of adrenal incidentalomas: Current status. AJR Am J Roentgenol 2002;179:559-68.
22. Kawashima A, Sandler CM, Ernst RD, Takahashi N, Roubidoux MA, Goldman SM, *et al*. Imaging of nontraumatic hemorrhage of the adrenal gland. Radiographics 1999;19:949-63.
23. Jordan E, Poder L, Courtier J, Sai V, Jung A, Coakley FV. Imaging of nontraumatic adrenal hemorrhage. AJR Am J Roentgenol 2012;199:W91-8.
24. Boland GW. Adrenal imaging: Why, when, what, and how? Part 2. What technique? Am J Roentgenol 2011;196:W1-5.
25. Blake MA, Kalra MK, Sweeney AT, Lucey BC, Maher MM, Sahani DV, *et al*. Distinguishing benign from malignant adrenal masses: Multi–detector row CT protocol with 10-minute delay. Radiology 2006;238:578-85.
26. Choi YA, Kim CK, Park BK, Kim B. Evaluation of adrenal metastases from renal cell carcinoma and hepatocellular carcinoma: Use of delayed contrast-enhanced CT. Radiology 2013;266:514-20.
27. Boland GW. Adrenal imaging: Why, when, what, and how? Part 1. Why and when to image? Am J Roentgenol 2010;195:W377-81.
28. Schwartz LH, Macari M, Huvos AG, Panicek DM. Collision tumors of the adrenal gland: Demonstration and characterization at MR imaging. Radiology 1996;201:757-60.
29. Paling M, Williamson B. Adrenal involvement in non-Hodgkin lymphoma. Am J Roentgenol 1983;141:303-5.
30. Rashidi A, Fisher SI. Primary adrenal lymphoma: A systematic review. Ann Hematol 2013;92:1583-93.
31. Mullins ME, Chao S, Dong H, Slanetz PJ. Post transplantation Non-Hodgkin's lymphoma of the adrenal gland. Am J Roentgenol 2001;177:336.
32. Singh D, Kumar L, Sharma A, Vijayaraghavan M, Thulkar S, Tandon N. Adrenal involvement in non-Hodgkin's lymphoma: Four cases and review of literature. Leuk Lymphoma 2004;45:789-94.
33. Elsayes KM, Mukundan G, Narra VR, Lewis JS Jr, Shirkhoda A, Farooki A, *et al*. Adrenal masses: MR imaging features with pathologic correlation. Radiographics 2004;24:S73-86.
34. Metser U, Miller E, Lerman H, Lievshitz G, Avital S, Even-Sapir E. 18F-FDG PET/CT in the evaluation of adrenal masses. J Nucl Med 2006;47:32-7.
35. Gupta P, Bhalla A, Sharma R. Bilateral adrenal lesions. J Med Imaging Radiat Oncol 2012;56:636-45.

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