

Adrenal Adenoma-Hemangioma Collision Tumor: Description of Two Cases

Michele Foresti^{1*}, Andrea Parmiggiani¹

1. Department of Diagnostic Imaging and Laboratory Medicine, Azienda USL-IRCCS di Reggio Emilia, Reggio Emilia, Italy

* **Correspondence:** Michele Foresti, Department of Diagnostic Imaging and Laboratory Medicine, Azienda USL-IRCCS di Reggio Emilia, via Amendola 2, Reggio Emilia, 42122, Italy
(✉ Michele.Foresti@ausl.re.it)

Radiology Case. 2019 Jun; 13(6):1-12 :: DOI: 10.3941/jrcr.v13i6.3691

ABSTRACT

Adrenal collision tumors are rare clinical entities referring to separate coexisting adjacent tumors involving an adrenal gland with sharp demarcation between the two and without a substantial histologic admixture at the interface. Most of the adrenal collision tumors described are combinations of adenoma and metastasis or adenoma and myelolipoma. We report two cases of a 63-year-old male and a 76-year-old female patient with a presumable exceedingly rare adrenal hemangioma-adenoma collision tumor. To our knowledge, only two reports of a collision tumor comprising an adrenal hemangioma and an adenoma have been described in literature.

CASE REPORT

CASE REPORT

Patient 1

A 63-year-old male presented to our institute in order to perform the 1st routine CT exam (Somatom Emotion 16 slice scanner, Siemens Medical Solutions, Forchheim, Germany), 6 months after an enucleoresection of a small left kidney clear cell carcinoma.

Preoperative exams like ultrasound, CT and MRI, as well as the enucleoresection itself, were performed in another institute and they were not available at the time of our 1st CT, which showed regular aspects of a kidney enucleoresection and a left adrenal oval lesion with smooth margins diagnosed in the other institute as a simple adrenal adenoma.

In our CT exam the adrenal lesion features were not completely suggestive of a simple adenoma: in the unenhanced sequence the lesion showed two different densities, one more peripheral with the classic adenoma features (density < 10HU) and another central one with a round morphology and with a

density of 25HU (Fig 1A). Arterial and venous phases showed faint inhomogeneities of the central component with the suspicion of little spots of globular enhancement at the interface between the two components (Fig 1B-1C). Delayed phase at 15minutes after contrast bolus injection showed a late enhancement of this central part of the lesion (Fig 1D).

Therefore, we performed a MRI exam (Siemens Magnetom Avanto 1,5 T Erlangen, Germany) in order to obtain a better characterization of this adrenal lesion: the peripheral component showed a classic adenomatous aspect with a signal loss in the out of phase T1w images, while the central one showed inhomogeneous hyperintensity in T2w and T2w fat sat images better seen at the interface (Fig 2A-2B), no signal restriction with high b value on DWI and an ADC value of $1299 \times 10^{-6} \text{mm}^2/\text{s}$ (Fig 2C-2D), isointensity compared with muscle and spleen in both in phase and out of phase T1w images (Fig 2E-2F) as well as in the unenhanced T1w 3D VIBE. The dynamic subsequent sequences showed a progressive centripetal globular enhancement till the complete persistent hyperintensity in the delayed phase at 15 minutes

after bolus administration (Fig 3A-3B-3C-3D-3E). Moreover, we retrospectively re-evaluated the preoperative MRI performed 6 months before our follow up CT exam and the adrenal lesion had the same features. On the basis of the overall benign features of this lesion, we postulated that the more probable diagnosis was an adrenal adenoma-hemangioma collision tumor and considering the wish expressed by the patient not to undergo another surgery or other invasive exams, a 6 months MRI follow up strategy has been established: after 24 months the lesion was unchanged as regards morphological and dynamic features (Fig. 4A-4B).

In this case, no functional investigations were performed regarding cortical or medullary overactivity since they were not considered necessary on the basis of the absence of a clinical expression that could suspect it.

Patient 2

A 76-year-old woman presented to our institution to perform a preoperative CT urography scan (Somatom Emotion 16 slice scanner, Siemens Medical Solutions, Forchheim, Germany) for a papillar bladder carcinoma to be removed and previously identified by ultrasound and diagnostic cystoscopy. The patient in the past underwent mastectomy for a breast cancer, pulmonary superior left lobectomy for an adenocarcinoma in 2013 and a lung segmentectomy for a MALT-oma in 2016.

At the basal CT examination, in addition to the bladder lesion, an oval 20mm nodule with regular margins was found in the left adrenal gland characterized by two components, a more peripheral one with a density lower than 10HU compatible with adenomatous tissue and a more central one with density of 28HU (Fig 5A). In the dynamic phases the central component had a globular enhancement, visible in the early arterial phase as small peripheral spots and then presenting a centripetal and progressive enhancement up to the persistent complete hyperdensity at about 15 minutes from the administration of the contrast medium (Fig 5B-5C-5D).

On ultrasound examination, this adrenal lesion appeared to be substantially hypoechoic with a central component that was slightly hyperechoic, corresponding to the non-adenomatous part (Fig 6).

Retrospectively analyzing some unenhanced thoracic follow up CT performed in previous years after lobectomy, we realized that this feature of the lesion was already visible and substantially unchanged even if considered purely adenomatous and therefore not further investigated (Fig 7A-7B).

Furthermore, by analyzing a previous MRI performed in 2013 for the study of the adrenal lesion identified in the context of the regular follow-up controls after mastectomy, the clearly adenomatous component was correctly identified and the more central, slightly hyperintense component was already identifiable in the sequences in phase opposition even though poorly appreciable (Fig 7C-7D).

In consideration of the morphological, densitometric and dynamic benignity characteristics of the lesion, above all the centripetal peripheral globular enhancement which, when present, was highly specific of hemangioma, we postulated that this lesion could be a case of adenoma-hemangioma adrenal collision tumor. Therefore, a clinical-radiological follow-up was simply set up also by virtue of the unmodified characteristics retrospectively seen from 2015 and the stability of the lesion 1 year after the CT urography. Also in this case, no functional investigations were performed regarding cortical or medullary overactivity since they were not considered necessary on the basis of the absence of a clinical expression that could suspect it.

DISCUSSION

Adrenal collisions tumors (ACTs) are defined as the coexistence of two adjacent, but histologically distinct tumors of the adrenal gland without a substantial histologic admixture at the interface. By contrast, composite tumors are neoplasms with an intimate admixture of two different cell types. The most commonly reported ACT comprises an adenoma and a myelolipoma. However, ACTs composed of adenoma and metastases (typically from lung and breast carcinomas and melanoma) are the most problematic in terms of diagnosis and appropriate patient management. In addition, various other ACTs, such as adenoma and pheochromocytoma or hemangioma, adrenocortical carcinoma and metastases or myelolipoma, adrenal carcinosarcoma and metastases, and myelolipoma and Hodgkin lymphoma, and other combinations have been described in literature, mostly as individual case reports [1,2,3,4,5,6,7,8,9]. Collision tumors are described in a variety of sites including lungs, bowels, genitourinary tract, meninges and lymph nodes other than adrenal glands [8].

Etiology & Demographics:

ACTs are rare tumors whose actual prevalence is unknown, despite the relatively high incidence of both benign and metastatic lesions of adrenal glands. The pathogenesis of ACTs has been debated, as the limited number of cases reported precludes detailed analysis of their etiology. Two theories have been postulated to describe their pathogenesis. The first and the simplest explanation is that two different primary tumors merely occur together by chance. A second hypothesis supposes that a single carcinogenic stimulus alters a particular region in the adrenal gland, allowing two separate tumors to occur in contiguity, or the presence of one tumor may alter the local environment, providing a fertile ground for the development of a second tumor [1].

Clinical & Imaging findings:

These tumors are usually incidentally detected during work-up for some other disease. In the adrenals, detection of incidentalomas has increased with the extensive use of cross-sectional imaging and most of these are adenomas or metastases. Preoperative diagnosis of individual components of ACTs is again another challenge [8].

Cross-sectional imaging studies such as CT, MRI, and PET/CT play an important role in the detection, characterization, and follow up of ACTs. In patients with potential ACTs, the goal of imaging is to differentiate the components with high specificity so that a metastatic focus is not mistaken for a benign lesion, thus potentially altering the treatment course and increasing patient morbidity. CT is often the first modality utilized in detecting adrenal masses. Attenuation values on unenhanced and contrast enhanced scans along with washout characteristics on delayed images are useful in differentiating potential benign and malignant components of an ACT. MRI is indicated for characterization of adrenal masses that show atypical findings on CT; images obtained with chemical shift imaging and gadolinium-enhanced techniques are most useful in delineating various tumor components [1]. If ACTs remain indeterminate on CT and MRI, [18F]fluorodeoxyglucose (FDG)-PET/CT can be performed to identify potential metabolically active foci within these tumors, which are generally considered to be malignant[10].

On CT, ACTs demonstrate imaging findings of two different neoplasms that show distinct pathologic features. An ACT should be considered if a heterogeneous adrenal lesion has appreciable differences in attenuation, or a previously known benign neoplasm changes in appearance or increases in size on follow-up imaging. These findings are particularly important in patients with known malignancies, and should raise a high suspicion for a potential ACT [1]. CT characterization of adrenal nodules can be performed without or with the use of iodinated contrast material. Unenhanced CT characterization of adrenal nodules relies on the detection of intracytoplasmic lipid found within adrenal adenomas, which reduces their CT attenuation. In lipid-rich adenomas, attenuation measurements will be less than 10 HU. This quantitative threshold is highly specific for the diagnosis of adenoma. In approximately one-third of adenomas, there is insufficient intracytoplasmic lipid content so that CT attenuation values will be greater than 10 HU (lipid-poor adenomas). Lipid poor adenomas can also be characterized using a dedicated adrenal washout CT protocol: at multiphase adrenal washout CT, adenomas show washout of contrast material over time; multiphase adrenal washout CT has been previously validated as a specific method to differentiate lipid-poor adenomas from metastatic disease using quantitative absolute and relative washout criteria [11]. In fact, they typically show relative percentage enhancement washout greater than 40% and absolute percentage washout greater than 60%. This phenomenon is due to the increased capillary permeability in malignant tumors, unlike adenomas with normal capillaries showing rapid contrast washout [12,13,14].

When results of CT examinations are equivocal, MR imaging is the next imaging study of choice for characterizing adrenal lesions. Various MR imaging parameters can be used to characterize adrenal masses, including T1 and T2 characteristics, calculated T2 values, enhancement patterns, and chemical shift characteristics. In general, metastases and carcinomas contain larger amounts of fluid than adenomas and thus appear bright on T2w images. However, there is significant overlap in T1 and T2 signal intensity between

adenomas and metastases, and thus signal intensity is not useful to reliably differentiate between them. Enhancement patterns have also been investigated as a means of differentiating benign adrenal adenomas from metastases, and, similar to their appearance at CT, adenomas vigorously enhance and exhibit early washout of contrast material compared with metastases on MR images. As stated earlier, intracellular lipid is high in most adrenal adenomas and low in metastases. Chemical shift imaging is an MR imaging technique used to detect lipid within an organ and is the most sensitive method for differentiating adenomas from metastases. In studies that have compared T1, T2, enhancement patterns, and chemical shift imaging for differentiating adenomas from metastases, the latter has demonstrated a high sensitivity and specificity. In fact benign adenomatous components show signal loss on opposed phase images, whereas metastatic components fail to show signal drop [15].

Although CT and MRI techniques can provide useful information on the anatomic details of ACTs, FDG-PET has the added advantage of providing a non-invasive technique of yielding functional characteristics of these tumors. A focus of increased FDG uptake in an ACT typically indicates the presence of malignancy, and allows the differentiation of malignant and benign components in ACTs. However, mild to moderately metabolically active adenomas may show increased FDG uptake and thus mimic malignancy, and hemorrhage and necrosis within a malignant lesion can be interpreted as benign. PET/CT is also not recommended for adrenal lesions smaller than 1 cm, as uptake in smaller lesions is typically less and cannot be accurately distinguished from normal tissue [1]. If the lesion is still uncertain with all these investigations then invasive methods like imaging-guided fine needle aspiration of the lesion or adrenal vein sampling may be tried and adrenalectomy is the last resort [8].

Adrenal hemangiomas are generally seen in people between the ages of 50–70, with a strong predilection for women. They are well encapsulated, originating from the adrenal cortex. On plain radiographs, punctate and stippled phlebolith-like calcifications may be seen. The phleboliths are better seen on CT. Large masses may demonstrate inhomogeneous density due to necrosis and fibrosis. Sonographic findings include a well circumscribed mass with heterogeneous echogenicity, often with anechoic areas and/or hyperechoic septa. Often the tumor is heterogeneous or cystic rather than solid. On MR, hemangiomas are generally hypointense on T1w and hyperintense on T2w images. Some may demonstrate central hyperintensity on T1w images corresponding to areas of hemorrhage. On both CT and MR, after the administration of intravenous contrast, hemangiomas typically demonstrate peripheral nodular enhancement with or without centripetal fill-in on delayed images. Centripetal enhancement is reportedly less frequent than in hepatic hemangiomas because of increased necrosis and fibrosis in adrenal hemangiomas. MRI findings of adrenal hemangiomas including T1 hyper or hypointensity and T2 hyperintensity are nonspecific and can be seen in other adrenal tumors such as pheochromocytomas, primary adrenal cortical carcinomas, and metastases. It has been suggested that centripetal enhancement on dynamic MRI has not been reported in other adrenal

tumors, and if present, should enable differentiation of hemangiomas from other adrenal tumors [9].

In our report, the adrenal lesions of both patients had substantially the same characteristics, and based on the densitometric, signal intensity and dynamic characteristics were more likely compatible with the hypothesis of adenoma-hemangioma collision tumors. The main limitation of our study consists in the absence of a histological finding that can confirm the diagnosis on the basis of imaging since in both cases the strategy chosen was that of radiological monitoring; therefore the diagnosis remains exclusively speculative. In both cases, however, the absolute stationary nature of the findings over time and the progressive centripetal dynamic behavior of the central component are the most important criteria that corroborate this hypothesis of adrenal adenoma-hemangioma.

Treatment & Prognosis:

The management of an adrenal collision tumor depends significantly on the components that compose it. If in fact at the end of the diagnostic path these components have benign characteristics, then they can tend towards a regular radiological follow up strategy; if, on the contrary, one of the two components or both have clinical, laboratory and radiological characteristics suspected for malignancy, then it can be operated by adrenalectomy in case of primitiveness or possibly chemotherapy and adrenalectomy in case of metastatic localization in selected patients. Additionally, the literature supports the possible benefit of resection of a solitary site of hematogenous metastasis [15]. The efficacy and safety of laparoscopic adrenalectomy has made metastasectomy a favored approach for patients with isolated metastatic disease. The laparoscopic approach is associated with shorter hospital stay, lower blood loss and lower morbidity. This, combined with the five-year survival data ranging from 24 to 31%, depending on the tumor type, makes the treatment approach reasonable in select patients [4].

Differential Diagnosis:

Considering that an ACT includes two separate different types of tumor, differential diagnosis relies on the imaging, clinical and laboratory features of each component of the ACT itself. Different components have been described in literature, some with pathognomonic features, some others with overlapping features and more difficult characterization. Besides adenomatous and hemangiomatous findings just described, other types of adrenal tumors included in the differential diagnosis are shown below.

Myelolipomas are uncommon benign lesions composed of mature adipose and hematopoietic tissue. The findings of a fatty mass at unenhanced CT is virtually diagnostic. Fat saturated MR imaging can help confirm the diagnosis by demonstrating signal dropout within the mass [16].

Hemorrhage: acute hemorrhage is characterized by the evolution of a non-enhancing low- or mixed-attenuation mass in one or both adrenal glands. In its mildest form, the peripheral distribution may account for the occasional observation of a train-track appearance of the adrenal gland

with preserved peripheral enhancement and central low attenuation. Other features that may be seen in acute adrenal hemorrhage include periadrenal infiltration, active extravasation with retroperitoneal bleeding, and maintenance of an adreniform shape. Unenhanced CT may show adrenal enlargement of greater than simple fluid attenuation and periadrenal infiltration. A hematoma appears on CT images as a circular non enhancing mass of greater than simple fluid attenuation (e.g., 50–90 HU). MRI may show high T1 signal intensity or rapidly evolving signal intensity. Over time, the size and CT attenuation of adrenal hemorrhage decrease, eventually reaching simple fluid attenuation or even complete resolution. MRI shows a hematoma as hyperintense on both T1w and T2w images from approximately 1 week to 2 months after trauma, after which the hematoma acquires a hypointense rim on both T1w and T2w images as the result of hemosiderin deposition and fibrosis. Later, chronic hemorrhage may appear as adrenal atrophy or a hemorrhagic adrenal pseudocyst. Atrophy appears on CT images as a shriveled, isoattenuating adreniform structure. A hemorrhagic adrenal pseudocyst is a chronic organized collection of hemorrhage that presents as non-enhancing, thin-rimmed cystic structures. At CT, pseudocysts are non-enhancing and have central hypoattenuation close to that of simple fluid. Most pseudocysts are unilocular and peripherally calcified. At MRI, the chronic presence of blood products can cause heterogeneous intracystic signal intensity. Calcifications are less well appreciated but can cause peripheral loss of signal intensity [16,17].

Pheochromocytomas: on unenhanced CT, pheochromocytomas have a varied appearance. They range from low density to soft tissue attenuation. Approximately two thirds of pheochromocytomas are solid and the rest are complex or cystic. Almost all pheochromocytomas have attenuation values of greater than 10 HU; however, rare intracellular fat-containing pheochromocytomas may result in attenuation values of less than 10 HU similar to adenomas. Alternatively, hemorrhage may increase the density of the pheochromocytoma. Calcifications are found in approximately 10% of pheochromocytomas. On contrast enhanced CT, pheochromocytomas may show homogeneous or variable enhancement, although most cases show avid enhancement of the solid components. Contrast washout in pheochromocytomas may be variable and may overlap with both benign lesions, such as adenomas, and malignant lesions. Earlier studies showed pheochromocytomas follow the washout characteristics of adrenocortical carcinomas (ACCs) and metastases with an absolute percentage washout of less than 60% and a relative percentage washout of less than 40% at 15 minutes. The classic imaging feature for pheochromocytomas is a “light-bulb” bright lesion on T2w imaging comparable to the signal intensity of cerebrospinal fluid. However, in reported case series, this classic appearance is variable in prevalence, ranging from 11% to 65% of pheochromocytomas. This variability is because of increased water content either in the cystic or in the liquefactive necrotic tumor. In approximately 35% of cases, pheochromocytomas may have low signal intensity on T2w imaging. On T1w imaging, pheochromocytomas are typically isointense to muscle and hypointense to liver. Appearances on T1w imaging

are quite variable if necrosis or hemorrhage is present. Pheochromocytomas typically show avid gadolinium enhancement, but enhancement can be variable depending on the presence of cystic-necrotic areas, which do not enhance. After the morphological imaging studies have been obtained, the functional imaging modality could be utilized to confirm the source of the increased production of catecholamines. For most of the cases one of the following methods is employed-123I-MIBG scintigraphy, 18F-FDG, or 18F-DOPA PET/CT and somatostatin receptor imaging [18,19].

Adrenocortical carcinomas: they are extremely rare, occurring in one of every 1 million individuals. Patients may complain of abdominal pain or present with a large, palpable mass. Approximately 30% of these tumors are hyperfunctioning, and Cushing syndrome may be evident at presentation. CT findings include a large (usually >5cm) mass with central areas of necrosis and hemorrhage and containing calcifications in 30% of cases [16]. They appear heterogeneous on both T1w and T2w images owing to the presence of internal hemorrhage and necrosis. Hemorrhagic byproducts, principally methemoglobin, can result in areas of high signal intensity within the lesion on T1w images; areas of necrosis have high signal intensity on T2w images. Adrenocortical carcinoma can contain foci of intracytoplasmic lipid, which results in a loss of signal intensity on out of phase images. Liver metastases, lymphadenopathy and venous invasion are usually present at time of diagnosis [20,21].

Metastases: metastases are the most common malignant lesions involving the adrenal gland. Adrenal metastases are found in up to 27% of patients with malignant epithelial tumors at autopsy. Common primary sites of tumors that metastasize to the adrenal glands include the lung, bowel, breast, and pancreas. Metastases are usually bilateral but may also be unilateral [20].

Intracellular lipid content of the adrenal mass represents the anatomic difference between adenomas and metastases, and differences in vascular enhancement patterns represent the physiologic difference. Adenomas have abundant intracytoplasmic fat in the adrenal cortex and thus have low attenuation at CT (threshold of 10HU). Conversely, metastases have little intracytoplasmic fat and thus do not have low attenuation at unenhanced CT. Although the finding of lower attenuation is useful to characterize an adenoma, up to 30% of adenomas do not contain sufficient lipid to have low attenuation at CT hence, a more useful parameter is the percentage of absolute washout of contrast material as just described [15].

Adrenal metastases usually exhibit low signal intensity on T1w images and high signal intensity on T2w images; on dynamic contrast enhanced MR images show early and intense enhancement with delayed washout [16]. The most important diagnostic feature is the lack of signal loss on out of phase images (in contradistinction to that seen with adrenal adenoma). When adrenal lesions cannot be accurately characterized with CT or MR imaging, PET and adrenal biopsy should be performed to establish a definitive diagnosis [20].

Radiology Case. 2019 Jun; 13(6):1-12

TEACHING POINT

Adrenal collision tumors are rare clinical entities referring to separate coexisting adjacent tumors with sharp demarcation between the two and without a substantial histologic admixture at the interface. Diagnostic imaging is very important in differentiating benign from malignant components of the collision tumor and the subsequent management. Adenoma-Hemangioma collision tumor is an exceedingly rare entity that should be considered in the differential diagnosis of a complex adrenal mass.

REFERENCES

1. Venkata S. Katabathina, Erin Flaherty, Ravi Kaza, Vijayanadh Ojili, Kedar N. Chintapalli, Srinivasa R. Prasad. Adrenal collision tumors and their mimics: multimodality imaging findings. *Cancer Imaging.* 2013; 13(4): 602-610. Published online 2013 Dec 30. PMID: PMC3893905.
2. Bertolini F, Rossi G, Focchi F, et al. Primary adrenal gland carcinosarcoma associated with metastatic rectal cancer: a hitherto unreported collision tumor. *Tumori* 2011; 97: 27e-30e.
3. Cai-Xiang Zhang, Ye Tian. Adrenal Collision Tumor Composed of Adrenocortical Adenoma and Pheochromocytoma. *Chin Med J (Engl)* 2018 Feb 5; 131(3): 374-375. PMID: PMC5798068.
4. Untch BR, Shia J, Downey RJ, Carrasquillo JA, Panicek DM, Strong VE. Imaging and management of a small cell lung cancer metastasis/adrenal adenoma collision tumor: a case report and review of the literature. *World J Surg Oncol.* 2014; 12: 45. Published online 2014 Feb 26. PMID: PMC3941693.
5. Dongyan Liu, Sahayini A. Kumar. An exceedingly rare adrenal collision tumor: adrenal adenoma-metastatic breast cancer-myelolipoma. *J Community Hosp Intern Med Perspect.* 2017 Oct; 7(4): 241-244. Published online 2017 Sep 9. PMID: PMC5637651.
6. Hye Seung Lee, Yoo Jin Choi, Chungyeul Kim, Baek-Hui Kim. Adrenal Collision Tumor: Coexistence of Pigmented Adrenal Cortical Oncocytoma and Ganglioneuroma. *Case Rep Surg.* 2016; 2016: 5790645. Published online 2016 Dec 8. PMID: PMC5178330.
7. Yulin Lai, Liang Zhou, Jia Hu, Wenhua Li, Lin Cui, Yongqing Lai, Liangchao Ni. Adrenal collision tumor (parachordoma and ganglioneuroma): A case report. *Mol Clin Oncol.* 2018 Jun; 8(6): 740-742. Published online 2018 Apr 19. Correction in: *Mol Clin Oncol.* 2018 Aug; 9(2): 238. PMID: PMC5958878.
8. Subhash Raveendran, Harish Babu SP, Arun Kumar ML et al. Adrenal Adenoma-Hemangioma: A Unique Collision Tumor. *World Journal of Endocrine Surgery,* Sept-Dec 2011;3(3):125-127.

9. Siddiqui AJ, Miller FH, Kasuganti, Nikolaidis P. Adrenal Hemangioma-Adenoma : An Exceedingly Rare Adrenal Collision Tumor. J Magn Reson Imaging 2009;29:949-952. PMID: 19306439.

10. Blake MA, Sweeney AT, Kalra MK, Maher MM. Collision adrenal tumors on PET/CT. AJR Am J Roentgenol 2004; 183: 864-865. PMID: 15344295.

11. Schieda N, Siegelman ES. Update on CT and MRI of Adrenal Nodules. AJR Am JRoentgenol 2017;208:1206-1217. PMID: 28225653.

12. Caoili EM, Korobkin M, Francis IR, Cohan RH, Dunnick NR. Delayed enhanced CT of lipid-poor adrenal adenomas. AJR Am JRoentgenol 2000; 175(5): 1411-1415. PMID: 11044054.

13. Caoili EM, Korobkin M, Francis IR, et al. Adrenal masses: characterization with combined unenhanced and delayed enhanced CT. Radiology 2002; 222: 629-633. PMID: 11867777.

14. Adrenal imaging with multidetector CT: evidence-based protocol optimization and interpretative practice. Johnson PT, Horton KM, Fishman EK. Radiographics 2009 Sep-Oct;29(5):1379-31. PMID: 19755598.

15. Mayo-Smith WW, Boland GW, Noto RB, Lee MJ. State-of-the-art adrenal imaging. Radiographics Jul-Aug;21(4):995-1012. PMID: 11452074.

16. Khati NJ, Javitt MC, Schwartz AM. Adrenal adenoma and hematoma mimicking a collision tumor at MRI imaging. Radiographics 1999 Jan-Feb; 19(1):235-259. PMID: 9925401.

17. Jordan E, Poder L, Courtier J, Sai V, Jung A, Coakley FV. Imaging of nontraumatic adrenal hemorrhage. AJR Am J Roentgenol. 2012 Jul;199(1):W91-98. PMID: 22733936.

18. Leung K, Stamm M, Raja A, Low G. Pheochromocytoma: the range of appearances on ultrasound, CT, MRI and functional imaging. AJR Am J Roentgenol 2013 Feb;200(2):370-378. PMID: 23345359.

19. Ctvrtlik F, Koranda P, Schovanek J, Skarda J, Hartmann I, Tudos Z. Current diagnostic imaging of pheochromocytomas and implications for therapeutic strategy. Exp Ther Med 2018Apr;15(4):3151-3160. PMCID: PMC5840941.

20. Elsayes KM, Mukundan G, Narra VR, Lewis JS jr, Shirkhoda A, farooki A, Brown JJ. Adrenal masses: mr imaging features with pathologic correlation. Radiographics 2004 Oct;24 Suppl 1:S73-86. PMID: 15486251.

21. Slattery JM, Blake MA, Kalra MK et al. Adrenocortical carcinoma: contrast wash out characteristics on CT. AJR Am J Roentgenol 2006 Jul;187(1):W21-24. PMID: 16794135.

FIGURES

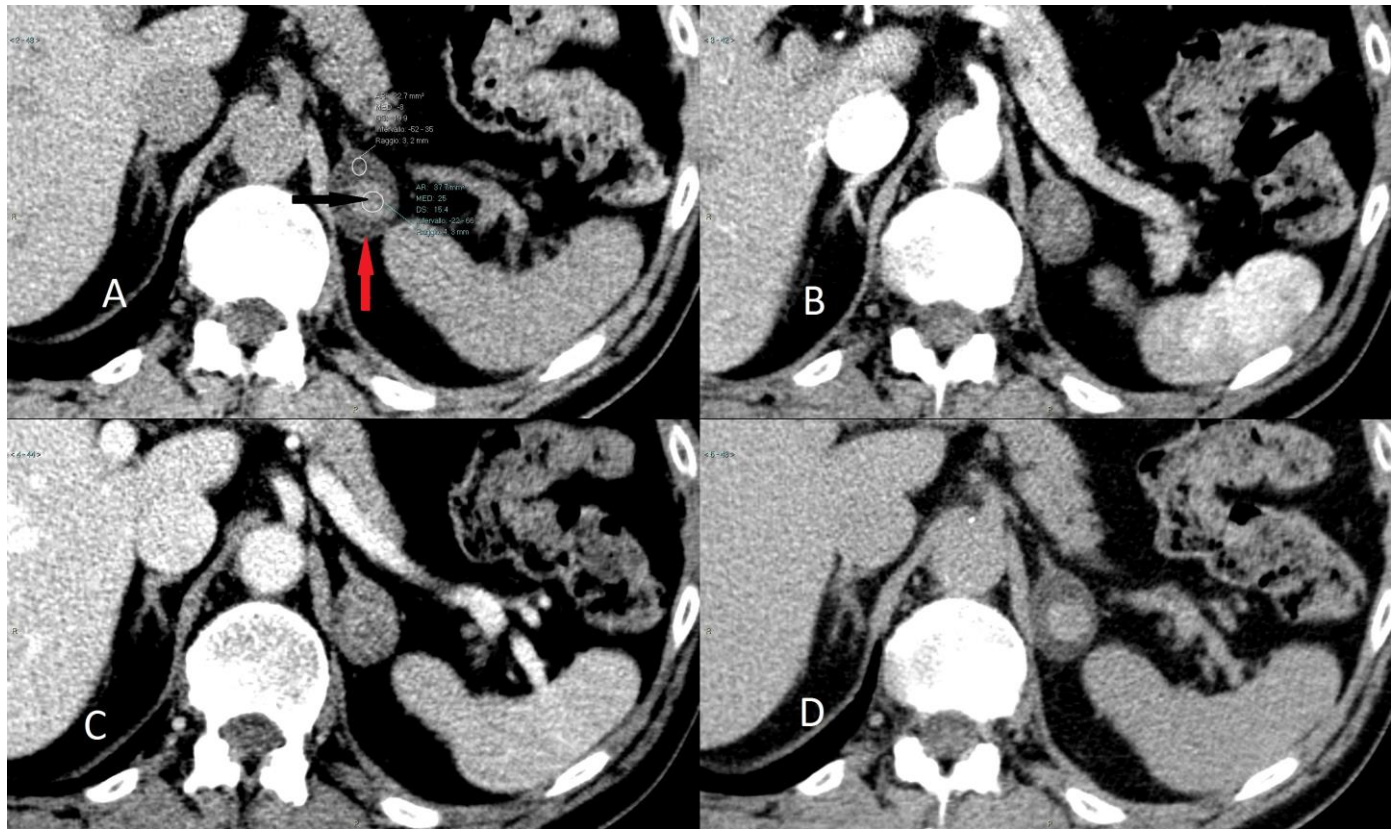


Figure 1: A 63-year-old man with an adenoma-hemangioma adrenal collision tumor.

Findings: in the unenhanced series the lesion shows two different densities, one more peripheral with the classic adenoma features and density < 10HU (red arrow) and another central one (black arrow) with a round morphology with a density of 25HU (A). Arterial (B) and venous (C) phases show faint heterogeneity of the central component with the suspicion of little globular enhancement points at the interface between the two components. Delayed phase at 15minutes after contrast bolus injection shows a late enhancement of this central part of the lesion, compatible with the hemangioma (delayed D).

Technique: Somatom Emotion 16 slice scanner, Siemens Medical Solutions, Forchheim, Germany. CARE Dose for automatic exposure control for tube voltage (kV) and effective tube current (mA), slices acquired with 3 mm slice thickness, 1 pitch, and 0.6 second rotation time. Contrast material was Iomeron® 350 (100 ml - 3ml/sec). Arterial phase acquired 35 seconds after bolus injection , venous phase after 80 seconds and delayed phase after 15 minutes.

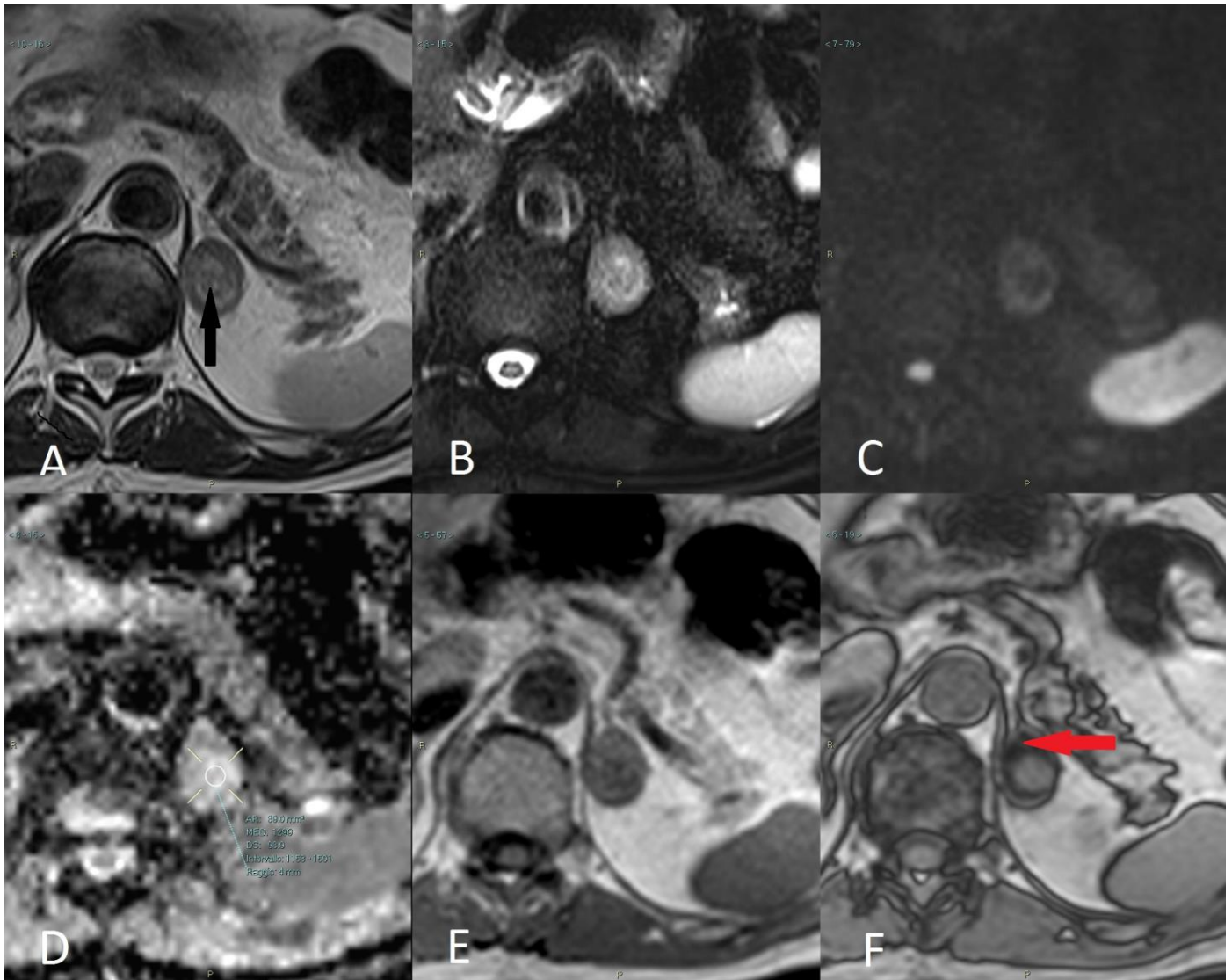


Figure 2: A 63-year-old man with an adenoma-hemangioma adrenal collision tumor.

Findings: the central component of the adrenal lesion (black arrow) shows heterogeneous hyperintensity in T2w and T2w fat sat images better seen at the interface (A-B), no signal restriction in diffusion weighted images (C-D), isointensity compared with muscle and spleen in both in phase (E) and out of phase T1w images (F) which on the contrary demonstrate the classic signal drop out in phase opposition of the peripheral adenomatous component (red arrow).

Technique: Siemens Magnetom Avanto 1,5 T Erlangen, Germany. T2w TSE 4mm slice thickness, TR 5913, TE 84 (A). T2 Haste fat sat 5mm, TR 1000, TE 98 (B). DWI 5mm, TR 6100, TE 83 and ADC map (C-D). T1w in phase 4 mm, TR 169, TE 4.92, FA 70 (E). T1w out of phase 4 mm, TR 169, TE 2.22, FA 70 (F).

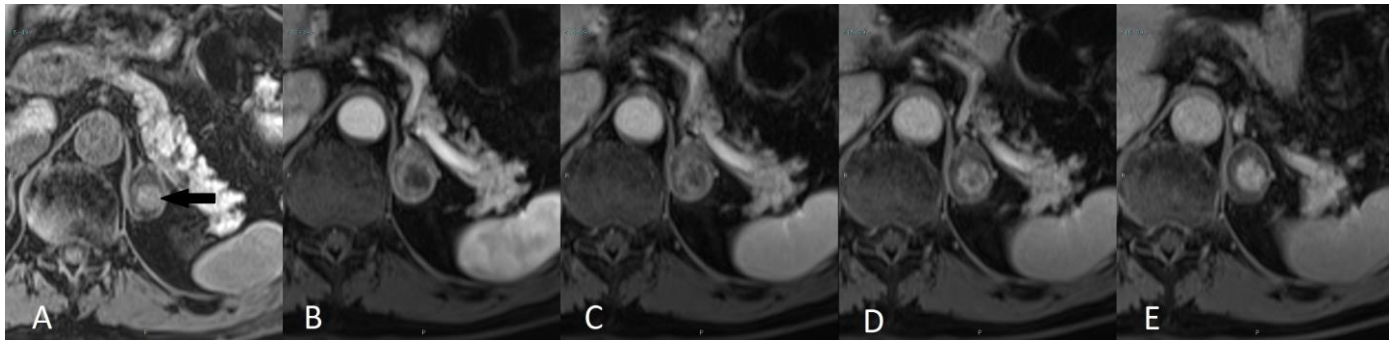


Figure 3: A 63-year-old man with an adenoma-hemangioma adrenal collision tumor.

Findings: the central component of the adrenal lesion (black arrow) shows isointensity compared with muscle and spleen in unenhanced T1w 3D VIBE (A). The dynamic subsequent sequences shows a progressive centripetal globular enhancement till the complete persistent hyperintensity in the delayed phase at 15 minutes after bolus administration compatible with hemangioma (arterial phase B - venous phase C - equilibrium phase D - delayed phase after 15 minutes E).

Technique: Siemens Magnetom Avanto 1,5 T Erlangen, Germany. VIBE 3D 2mm slice thickness TR 4.71, TE 2.39, FA 10. Contrast material was Dotarem® (14 ml - 2.5ml/sec).

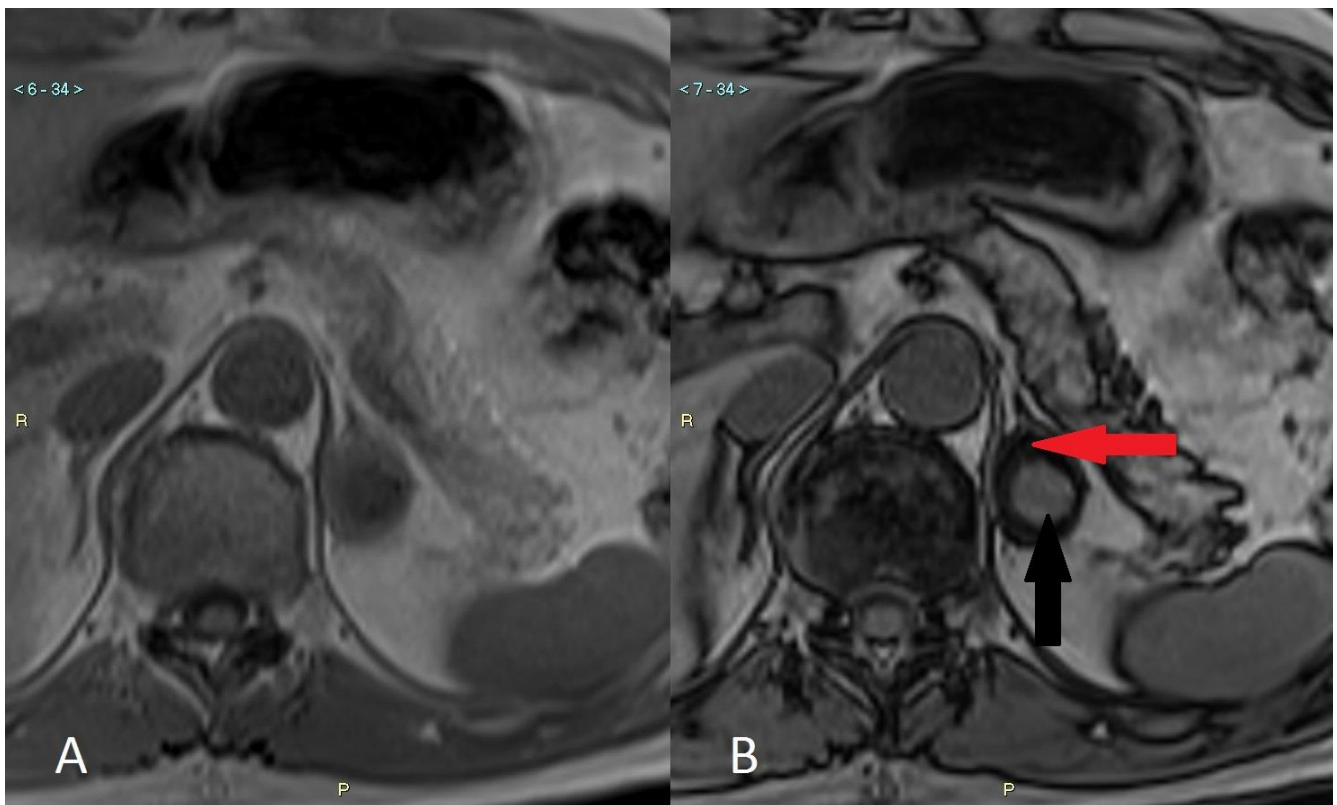


Figure 4: A 63-year-old man with an adenoma-hemangioma adrenal collision tumor. 2018 Magnetic Resonance Imaging (follow up exam after 24 months).

Findings: in phase (A) and out of phase T1w images (B) show unmodified findings of the left adrenal lesion. Central hemangiomatous component: black arrow. Peripheral adenomatous component: red arrow.

Technique: Siemens Magnetom Avanto 1,5 T Erlangen, Germany. T1w in phase 4 mm, TR 169, TE 4.92, FA 70 (A). T1w out of phase 4 mm, TR 169, TE 2.22, FA 70 (B).

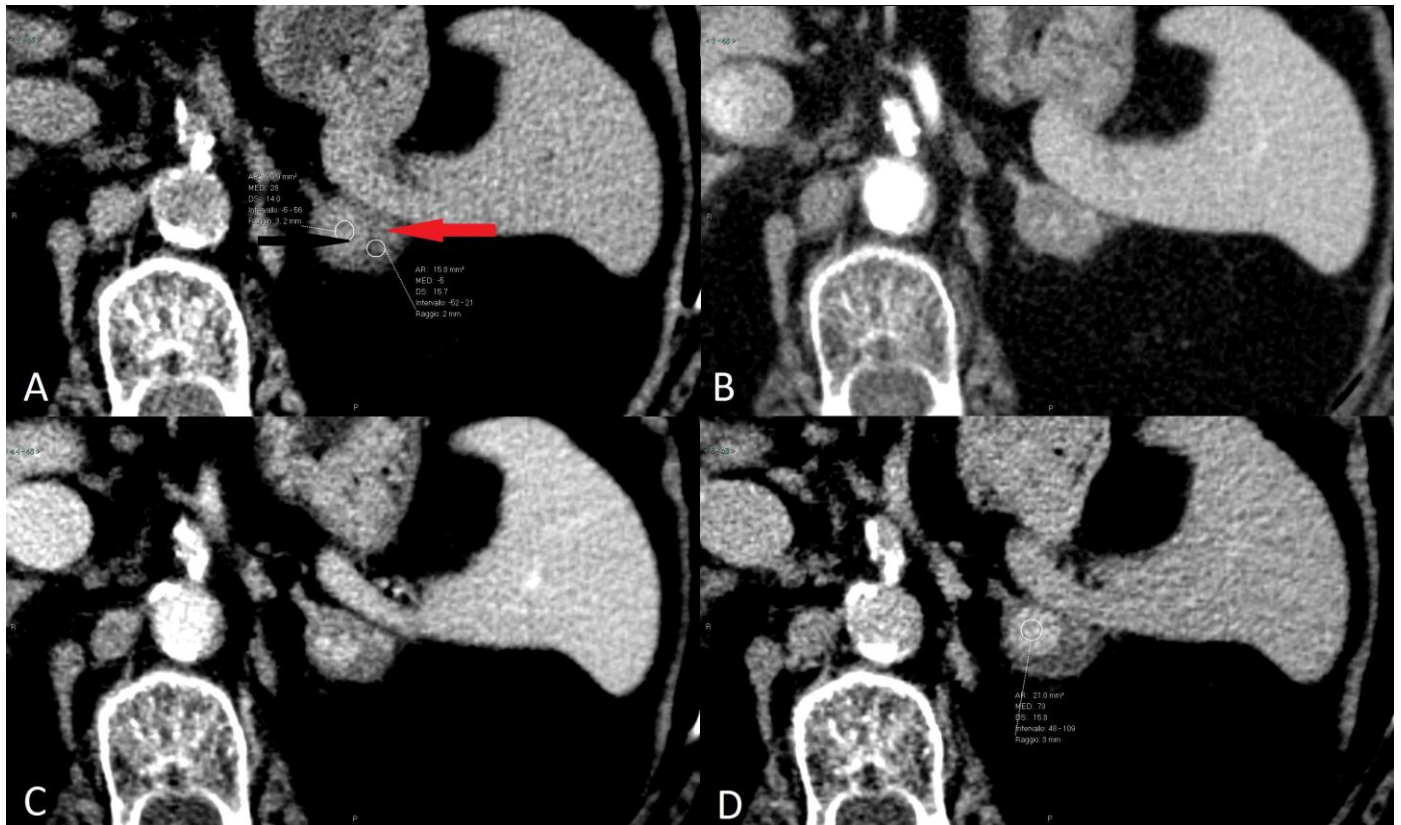


Figure 5: A 76-year-old woman with an adenoma-hemangioma adrenal collision tumor.

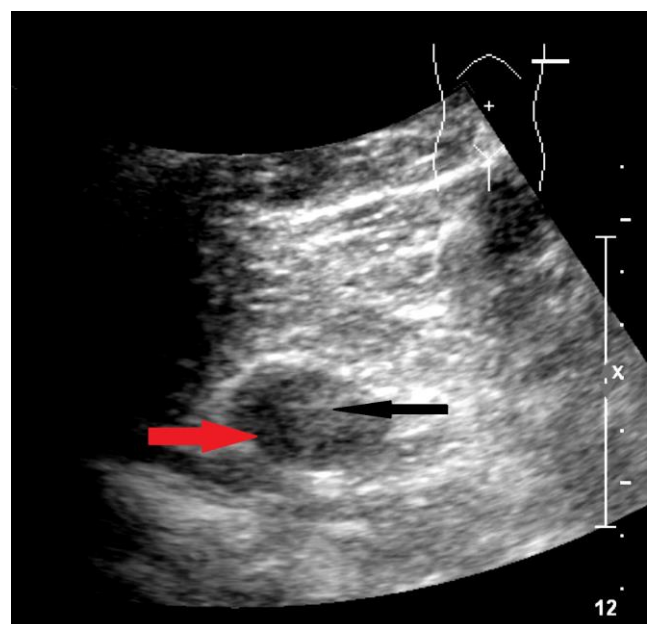
Findings: in the unenhanced series the lesion shows two different densities, one more peripheral with the classic adenoma features and density < 10HU (red arrow) and another central one (black arrow) with a round morphology with a density of 28HU (A). In the dynamic arterial (B) and venous (C) phases, the central component shows a globular enhancement, visible in the early arterial phase as small peripheral spots and then presenting a centripetal and progressive vascularization up to the persistent complete hyperdensity at about 15 minutes from the administration of the contrast medium (delayed D).

Technique: Somatom Emotion 16 slice scanner, Siemens Medical Solutions, Forchheim, Germany. CARE Dose for automatic exposure control for tube voltage (kV) and effective tube current (mA), slices acquired with 3 mm slice thickness, 1 pitch, and 0.6 second rotation time. Contrast material was Omnipaque® 350 (97ml - 3ml/sec). Arterial phase acquired 35 seconds after bolus injection, venous phase after 80 seconds and delayed phase after 15 minutes.

Figure 6 (right): A 76-year-old woman with an adenoma-hemangioma adrenal collision tumor.

Findings: this adrenal lesion appears to be substantially hypoechoic (red arrow) with a central component that is slightly hyperechoic, corresponding to the non-adenomatous part (black arrow).

Technique: Philips iU22 Ultrasound Machine. C5-2 MHz convex probe.



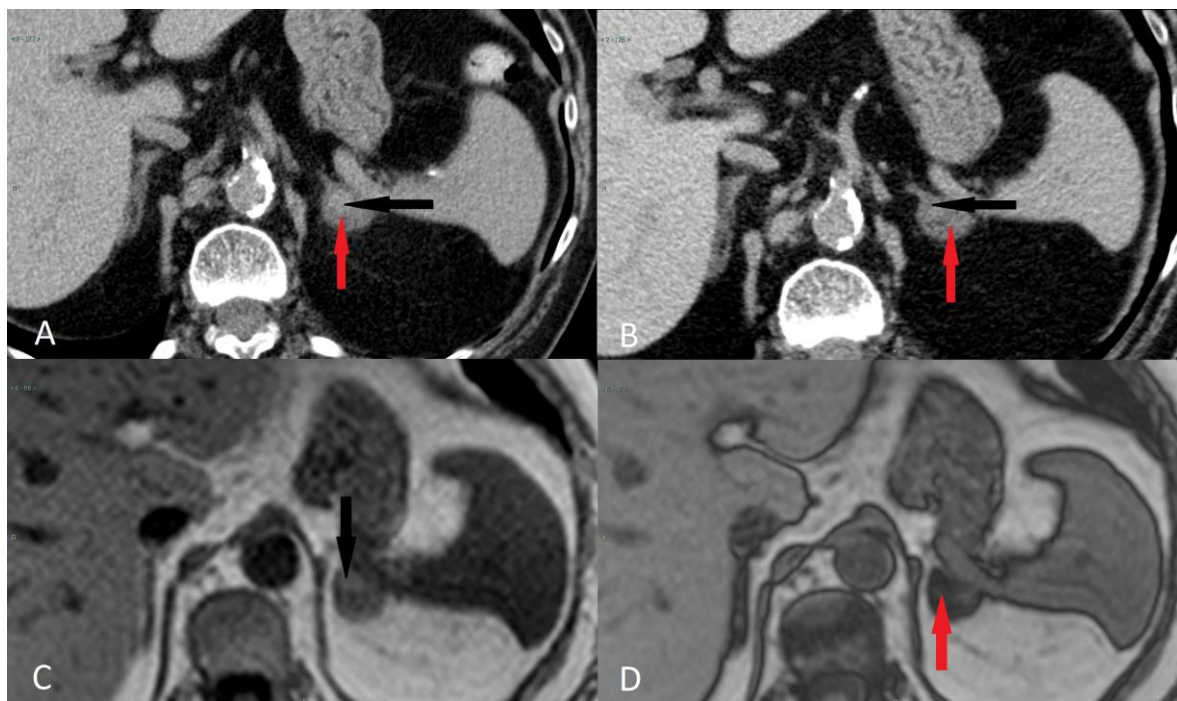


Figure 7: A 76-year-old woman with an adenoma-hemangioma adrenal collision tumor. 2016 Computed Tomography (A). 2015 Computed Tomography (B). 2013 Magnetic Resonance Imaging (C-D).

Findings: in unenhanced thorax CT performed respectively in 2016 (A) and 2015 (B) after lobectomy and retrospectively reviewed, both components of the adrenal lesion were already visible and substantially unchanged (central hemangioma component: black arrow. Peripheral adenomatous component: red arrow). Furthermore, even in a previous MRI performed in 2013, both the most central component characterized by tenuous hyperintensity in the T1w in phase sequence (black arrow in C) and the adenomatous component in the T1w out of phase sequence (red arrow in D), were already appreciable.

Technique: Somatom Emotion 16 slice scanner, Siemens Medical Solutions, Forchheim, Germany (A-B). CARE Dose for automatic exposure control for tube voltage (kV) and effective tube current (mA), slices acquired with 3 mm slice thickness, 1 pitch, and 0.6 second rotation time.

Siemens Magnetom Avanto 1,5 T Erlangen, Germany. T1w in phase 4 mm, TR 169, TE 4.92, FA 70 (C). T1w out of phase 4 mm, TR 169, TE 2.22, FA 70 (D).

Etiology	Debated. The first explanation is that two different primary tumors merely occur together by chance. A second hypothesis supposes that a single carcinogenic stimulus alters a particular region in the adrenal gland, allowing two separate tumors to occur in contiguity.
Incidence	Collision tumors are very rare. Adenomas are the most common adrenal lesions, found in 3% of cases at autopsy.
Gender Ratio	Unknown as far as concerned the collision tumor. Adrenal hemangiomas are generally seen with a strong predilection for women.
Age predilection	Unknown as far as concerned the collision tumor. Adrenal hemangiomas are generally seen in people between the ages of 50–70. Adrenal adenomas are found in almost all age groups increase in frequency with age.
Treatment and prognosis	It depends on the presence of only benign lesions or the presence of a malignant one. In the former case an imaging follow up management can be established. If there is a malignant component every case must be evaluated, and strategies can be chemotherapeutic or surgical. Most guidelines recommend excision if an adrenal lesion is >4 cm.
Findings on imaging	Each component of the collision tumor presents its own features. - Adrenal adenomas most important findings: density inferior to 10HU in unenhanced CT, absolute wash out more than 60%, and signal drop in out of phase T1w sequences. - Hemangiomas: inhomogeneous density at unenhanced CT due to cystic component, hemorrhage or calcifications. Hypointensity in T1w and hyperintensity in T2w sequences. On both dynamic CT and MR hemangiomas typical peripheral nodular enhancement with or without centripetal fill-in on delayed images. Centripetal enhancement on dynamic MRI has not been reported in other adrenal tumors, and if present, should enable differentiation of hemangiomas from other adrenal tumors.

Table 1: Summary table for adenoma - hemangioma adrenal collision tumor.

	CT	MRI	PET
Myelolipoma	Adipose density components.	Adipose components with signal drop in stir or fat saturation T1-T2w sequences.	No FDG uptake
Hemorrhage	<ul style="list-style-type: none"> Acute: circular non enhancing mass of greater than simple fluid attenuation. Over time, the size and CT attenuation decrease, eventually reaching simple fluid attenuation or even complete resolution. 	<ul style="list-style-type: none"> Hyperintense on T1w and T2w images from approximately 1 week to 2 months after trauma, after which the hematoma acquires a hypointense rim on both T1w and T2w images as the result of hemosiderin deposition and fibrosis. Chronic hemorrhage may appear as adrenal atrophy or a hemorrhagic adrenal pseudocyst. 	No FDG uptake
Pheochromocytoma	<ul style="list-style-type: none"> Unenhanced CT: they range from low density to soft-tissue attenuation. Two thirds of pheochromocytomas are solid and the rest are complex or cystic. Attenuation values of greater than 10 HU. Hemorrhagic foci or calcifications. CECT: homogeneous or variable enhancement, although most cases show avid enhancement of the solid components. They usually show an absolute percentage washout of less than 60%. 	<ul style="list-style-type: none"> “Light-bulb” bright lesion on T2w imaging. On T1w imaging typically isointense to muscle and hypointense to liver. Appearances on T1w imaging are quite variable if necrosis or hemorrhage is present. Avid gadolinium enhancement. Cystic-necrotic areas do not enhance. 	<ul style="list-style-type: none"> F-18 FDG PET/TC positive Ga-68 DOTATATE PET/CT positive F-18 DOPA PET/TC I-123 MIBG scintigraphy positive
Adrenocortical carcinoma	<ul style="list-style-type: none"> Usually a large mass with central areas of necrosis and hemorrhage Containing calcifications in 30% of cases. 	<ul style="list-style-type: none"> Heterogeneous on both T1-T2w images owing to the presence of internal hemorrhage and necrosis. Methemoglobin can result in areas of high signal intensity within the lesion on T1w images; areas of necrosis have high signal intensity on T2w images. Intracytoplasmic lipid results in a loss of signal intensity on out-of-phase images. 	F-18 FDG PET/TC positive
Metastasis	<ul style="list-style-type: none"> Little intracytoplasmic fat and thus do not have low attenuation at non-enhanced CT. Absolute wash out is usually inferior to 60%. 	<ul style="list-style-type: none"> Hypointense on T1w images. Hyperintense on T2w images. Early and intense enhancement, progressive with delayed washout. Lack of signal loss on out-of-phase images. Restriction in DWI. 	F-18 FDG PET/TC positive

Table 2: Differential diagnoses table for adenoma - hemangioma adrenal collision tumor.

ABBREVIATIONS

- ACT(s): Adrenal Collision Tumors
- CT: Computed Tomography
- DWI: Diffusion weighted imaging
- Fat sat: Fat saturation
- HU: Hounsfield Unit
- MRI: Magnetic Resonance Imaging
- T1w: T1 weighted
- T2w: T2 weighted
- VIBE: Volumetric interpolated breath-hold examination

KEYWORDS

adrenal collision tumor; adrenal adenoma; adrenal hemangioma; Magnetic Resonance Imaging; Computed Tomography

Online access

This publication is online available at:
www.radiologycases.com/index.php/radiologycases/article/view/3691

Peer discussion

Discuss this manuscript in our protected discussion forum at:
www.radiolopolis.com/forums/JRCR

Interactivity

This publication is available as an interactive article with scroll, window/level, magnify and more features.
 Available online at www.RadiologyCases.com

Published by EduRad



www.EduRad.org