# Anastomosing Hemangioma Incidentally Found in Kidney or Adrenal Gland: Study of 10 Cases and Review of Literature

Jun Zhou[1]*, Xiaoqun Yang[1]*, Luting Zhou[1],Ming Zhao[2], Chaofu Wang[1]

**Purpose:** To describe and report a series of renal and adrenal anastomosing hemangioma (AH) and to investigate its distinctive clinicopathologic features and review its clinical data available in the literature.

**Materials and Methods:** Clinical data of 10 AHs were retrospectively studied. Imaging and histologic features were re-evaluated and summarized. Immunostaining markers performed include CD31, CD34, ERG, Fil-1, D2-40, AE1/AE3, SMA, CD10, HHV8, S100, Ki-67. A follow-up of all cases was performed. Other AHs published in PubMed and Web of Science were reviewed.

**Results:** All of 10 AHs were found incidentally in 5 female and 5 male patients (median age, 48.5 years; mean, 51.7 years) and involved unilateral kidney (n=7) and adrenal glands (n=3) respectively. All lesions were well-defined in imaging and histologic examination. AHs were morphologically characterized by prominent anastomosing vascular channels without evidence of infiltration to surrounding normal tissues and significant cellular atypia. CD31, CD34, ERG were positive and Ki-67 showed typically low positivity (< 3%). All Patients underwent a mass resection and none of them had evidence of recurrence. Together with other cases published, the AHs showed distinctive clinicopathologic features with an excellent prognosis.

**Conclusion:** Renal or adrenal AH is a very rare vascular tumor. They have distinctive histologic features and a favorable prognosis. It is frequently mimicking well-differentiated angiosarcomas which easily results in unnecessary overtreatment in clinical practice.

**Keywords:** anastomosing hemangioma; clinicopathology; prognosis; kidney; adrenal gland

## INTRODUCTION

Vascular tumors are large and heterogeneous entities with varying endothelial differentiation.[1] Their complex categorization and clinical-biologic behaviors are usually not familiar to the clinicians.[2] So are the pathologists due to the broadly morphologic and relevant little genetically available information for the majority of vascular lesions and hence the differential diagnosis could be pretty challenging.[3] Primary benign vascular lesions arising in the kidney or adrenal gland are distinctly rare, among which arteriovenous malformation, angiomatous endothelial cysts, hemangioma, and angiosarcoma are the most common types.[4,5] Anastomosing hemangioma (AH), a very rare vascular tumor and was firstly described by Montgomery and Epstein in 2009.[6] One issue of concern for AH is mimicking malignancy histologically that may result in unnecessary overtreatment. Although AH was originally considered to be a distinct vascular tumor exclusively occurring in the genitourinary tract, the following reports indicated they can develop in various anatomic locations such as soft tissue, liver, colon, bladder, skin, etc.[7-10] Although some renal and adrenal AHs were sporadically reported, most pathologists and clinicians are still unfamiliar with this entity. Herein, we added 10 AHs arising in the kidney or adrenal gland AH to further investigate their clinicopathologic features and emphasize its diagnostic pitfalls and mimickers.

## MATERIALS AND METHODS

### *Subjects*

This study was approved by the Institutional Review Board at the Department of Pathology, Ruijin Hospital, Shanghai Jiaotong University School of Medicine (Shanghai, China). A total of 10 patients specimens entered our study, including 2 consultant cases and 8 surgical specimens. All of them have complete clinical data. All hematoxylin and eosin (HE)-stained (DAKO

[1]Department of Pathology, Ruijin Hospital, Shanghai Jiaotong University School of Medicine Shanghai, China.
[2]Department of Pathology, Zhejiang Provincial People's Hospital, Hangzhou, Zhejiang, China.
* J. Z. and X. Y. contributed equally.
*Correspondence: Department of Pathology, Ruijin Hospital, Shanghai Jiaotong University School of Medicine, No. 197 Ruijin Er Road, Huangpu District, Shanghai, 200025, China,
Tel: +86 17321086332, E-mail: wangchaofu@126.com.
*Department of Pathology, Ruijin Hospital, Shanghai Jiaotong University School of Medicine, No. 197 Ruijin Er Road, Huangpu District, Shanghai, 200025, China,
Tel: +86 17701879546, E-mail: yangxiaoqun963@163.com.
Received May 2020 & Accepted August 2020

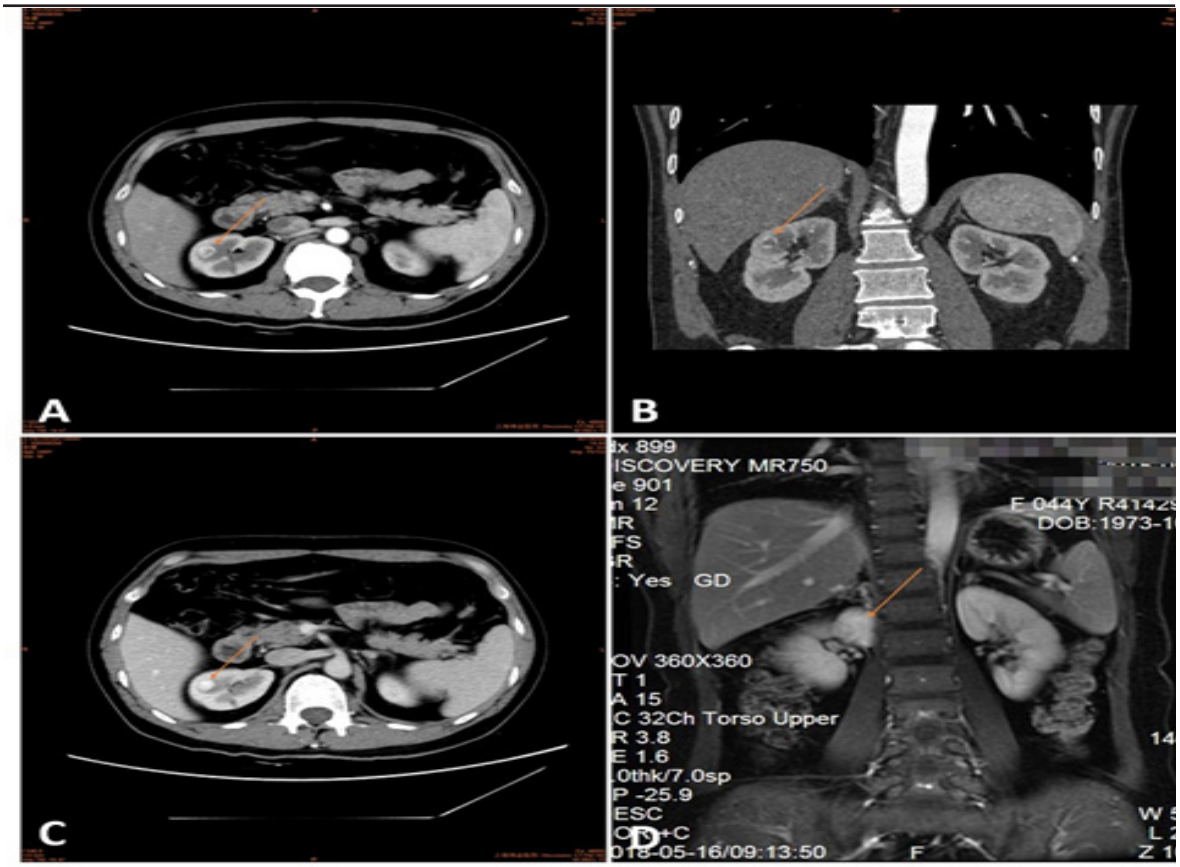


**Figure 1.** Represented imaging features of AH. A contrast-enhanced CT scan showed a well-circumscribed solid tumor in the right kidney with peripheral enhancement at the arterial phage (**A.** axial; **B.** coronal), and homogeneous enhancement at portal phase **(C)**. Coronal enhanced CT demonstrated a homogeneous enhanced lesion arising on the adrenal gland at arterial phage **(D)**.

CoverStainer; Agilent, Santa Clara, CA, USA) slides were independently reviewed by 2 experienced pathologists (C.F.W. and J. Z.) under a light microscope (BX43; Olympus Corporation, Tokyo, Japan). Follow-up was performed in the office setting or by telephone interview.

### *Immunohistochemistry (IHC)*

IHC evaluation of consultant cases was recorded according to the submitted immunostaining slides. Each surgical specimen was specially re-sectioned. 4-μm thick sections were taken from 10% formalin-fixed and paraffin-embedded tissue blocks followed by immunohistochemical staining using commercially available antibodies as follows: CD31 (monoclonal, prediluted; Dako, Glostrup, Denmark ), CD34 (QBEND, prediluted; Dako, Glostrup, Denmark), ERG (EP111, 1:200 dilution; ZSGB-BIO, Beijing, China), Fil-1(MRQ-1, 1:50 dilution; ZSGB-BIO, Beijing, China), D2-40 (D2-40, prediluted, Dako, Carpinteria, CA, US), AE1/AE3 (AE1/AE3, prediluted; Dako, Carpinteria, California, USA), SMA(1A4, 1:200; ZSGB-BIO, Beijing, China), CD10(56C6, 1:100 dilution; Leica Biosystem, Newcastle, UK), HHV8(LN35, 1:100 dilution; Abcam, Hong-Kong, China), S100 (Polyclonal, prediluted; Dako, Glostrup, Denmark), and Ki-67 (MIB-1, prediluted; Dako, Glostrup, Denmark). Detection of antibody binding was obtained using the universal immunoperoxidase polymer method (Envision-kit; Dako, Carpinteria, CA, USA). A Dako automated immunohistochemistry system (Dako, Carpinteria, CA, USA) was performed according to the manufacturer's protocol. The IHC results were independently interpreted by 2 experienced pathologists (L. Z. and C.F.W.).

### *Literature review and data analysis*

Literature in PubMed and Web of Science before April 2019 were reviewed. Only the cases that met the diagnosis of AH and had complete clinicopathologic and follow-up data entered our analysis. Descriptive statistics of the common features of AHs (epidemiology, demography and clinical features) were performed using the MS-office 2016 Excel software (Microsoft, Redmond, WA, USA).

## RESULTS

### *Clinical features and follow-up*

As a regional consultation and treatment center, the patients in our group mainly came from the eastern China during April 2015 to January 2019. The main clinical data of AHs are summarized in Table 1. A total of 8 renal and 2 adrenal lesions in 5 females and 5 males, ranging in age from 28 to 71 years (median, 48.5 years; mean, 51.7 years) were investigated. All the patients were asymptomatic and laboratory test results did not show significant abnormalities. Each lesion was accidentally found by routine physical examination, 1 of which was appreciated by imaging before her preoperative preparation for hepatic carcinoma. All lesions

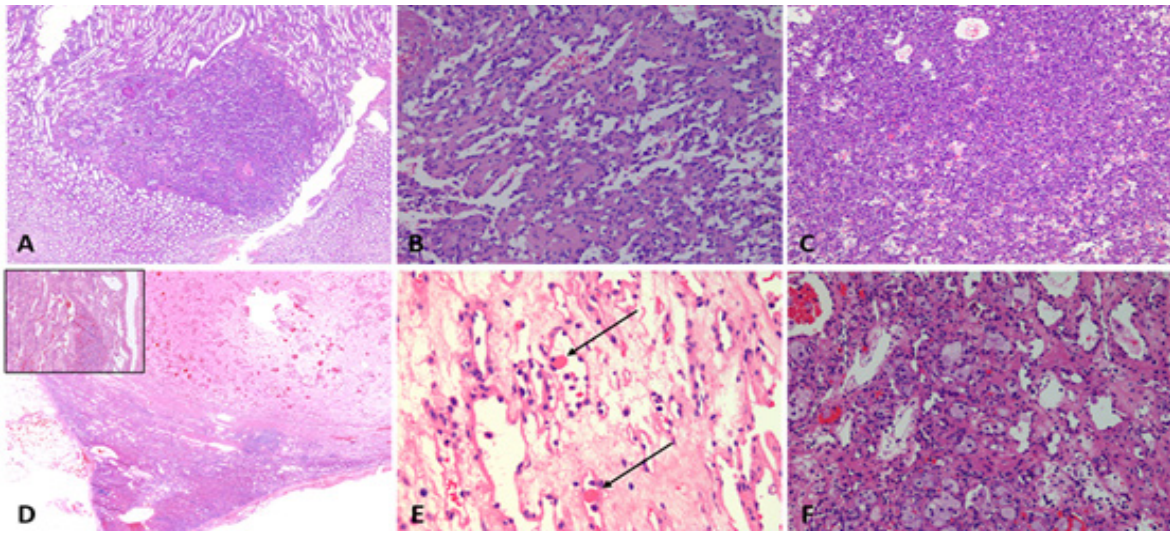


**Figure 2.** Histologic features of AH. Low power showed a well-delineated lesion developing in the renal parenchyma (**A**, staining H&E; staining H&E; magnification, 40×). At medium power, AH was characterized by prominent anastomosing vascular lining by singly bland endothelial cells (**B**, staining H&E; magnification, 200×). Cellular regions were a florid proliferation of interconnecting capillary-like vessels (**C**, staining H&E; magnification, 100×). This adrenal AH showing distinct loosely edematous stroma may obscure the anastomosing growth pattern (D, staining H&E; magnification, 40×), however, the typical growth areas can be focally appreciated (inset). eosinophilic granular materials (E, staining H&E; magnification, 200×), or foamy cells (F, staining H&E; magnification, 200×) was occasionally seen.

demonstrated a blurred mass with a soft-tissue density in computed tomography (CT), 8 cases showed peripheral enhancement at arterial phage and homogeneous enhancement at portal phase (**Figure 1A-C**); 2 cases directly displayed diffuse even enhancement at arterial phage (**Figure 1D**). All AHs were generally small, ranging in size from 0.8 to 3.9 cm (mean, 2.0 cm; median, 2.0 cm). All follow-up data were available, showing no evidence of recurrence (average follow-up 33 months, range 10 to 46 months).

Pathologic features

Grossly, the tumors imparted gay-red or purple-red solid appearance. Microscopically, all AHs were well-circumscribed and unencapsulated (**Figure 2A**). They typically consisted of anastomosing capillary-like vascular channels with no or mild atypia of tumor cells (**Figure 2B**). Mitosis was consistently absent. Alternating cellular (**Figure 2C**) and acellular regions were readily apparent in 4 cases (cases 1, 2, 8 and 9). The acellular areas were characterized by loosely edematous or hyaline stroma (**Figure 2D**). Proliferated vessels formed the glomerulus-like structure in 2 cases (cases 1, and 6). Case 10 had focally and centrally fibrosis. Extramedullary hematopoiesis (case 2), extracellular eosinophilic granular materials (case 1, **Figure 2E**), or foamy cells (case 4, **Figure 2F**) were also seen.

Immunohistochemically, tumor cells of all the cases were consistently positive for vascular endothelial markers including CD31, CD34 (Figure 3A), ERG (Figure 3B), and Fli-1. None of the cases were immunoreactive for AE1/AE3, EMA, PAX8, CD10, SMA, HHV8, S100, and D2-40. The proliferative index labeled by Ki-67 was typically low (range, 1%-4; mean, 2.2%; **Figure 3C**).

### *Clinical features summarized from publication*

Almost all AHs in the available literature were included to summarize the clinicopathologic features. (4,6-34) As depicted in Figure 4A, the 131 cases AHs (including our 9 cases) mostly reported from North America

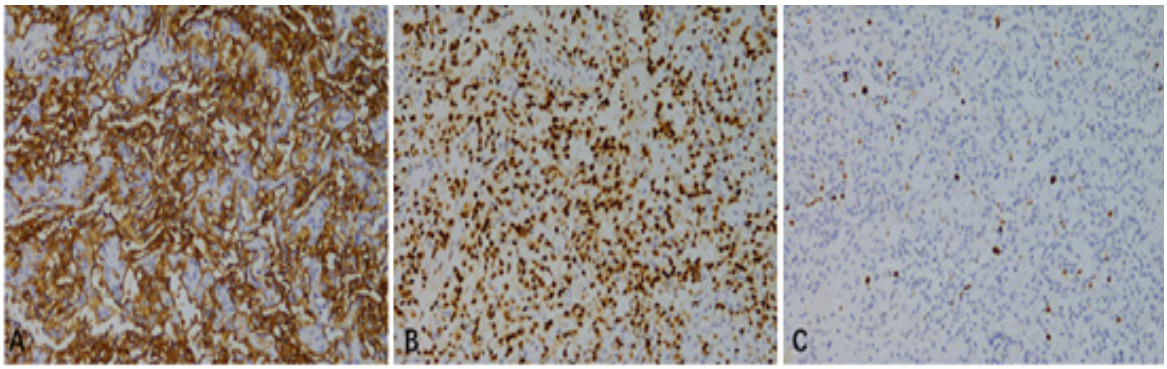


**Figure 3.** Immunophenotype of AH. The tumor cells expressed CD34 (**A;** magnification, 200×) and ERG (**B;** magnification, 200×), with a low proliferated index Ki-67 (**C;** staining **H&E;** magnification, 100×).

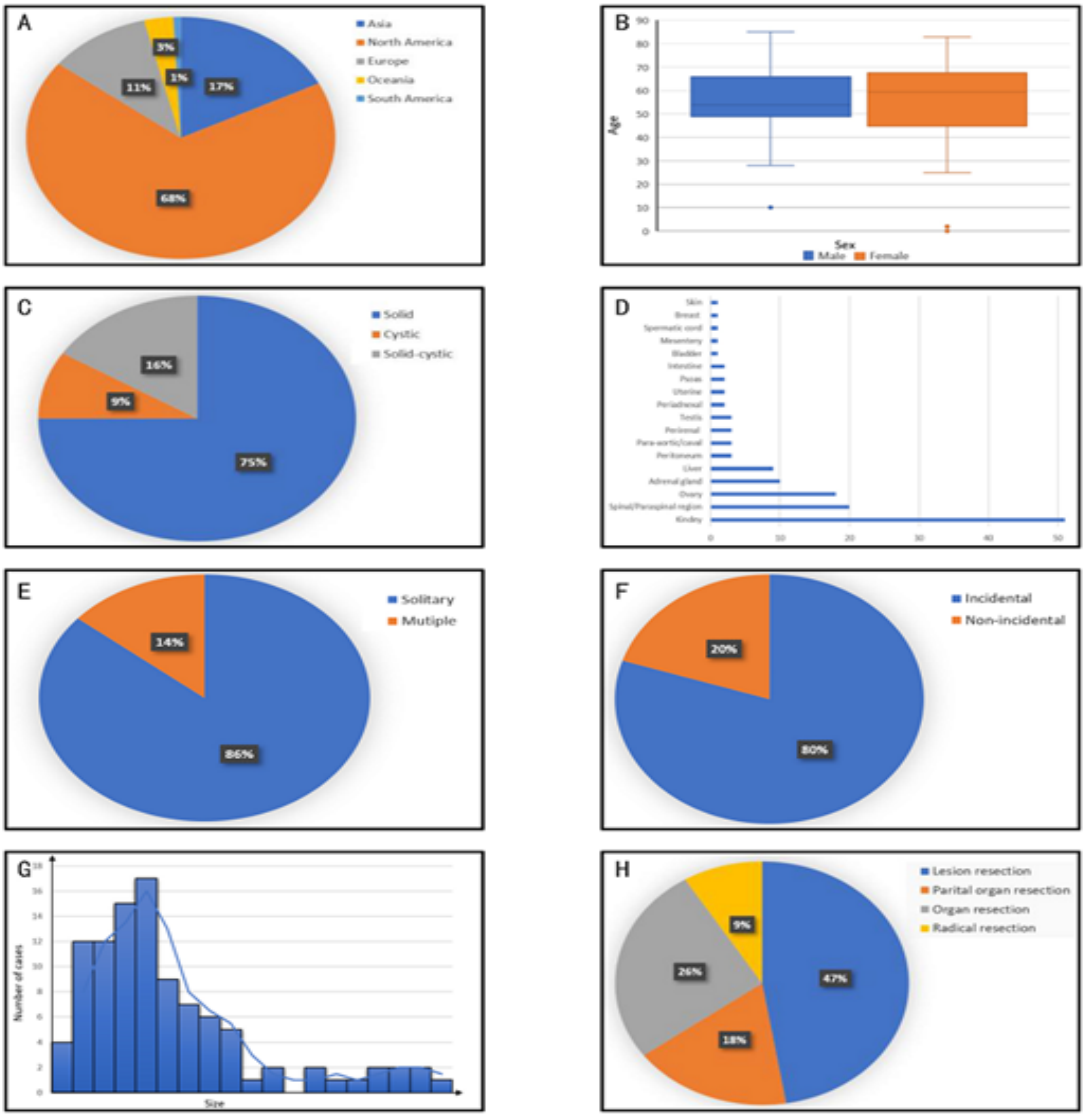


**Figure 4.** Clinical data of AH from literature. The cases reported from a distinct region around the world, most of which derived from North America **(A).** The female average age is slightly older than male **(B)**. Most of the tumors were incidentally found **(C)**. Various involved locations were reported as yet, of which the most location was kidney **(D)**. The majority demonstrated solitary lesion, but a subset of multiple lesion can also be appreciable **(E)**. The texture of AH can be solid, cystic and solid-cystic **(F)**. The tumors ranged in size from 0.5 to 12 cm, most of which clustering in 0.5 to 3.5 cm **(G)**. The surgical method includes simple lesion resection, organ resection, partial organ resection, radical resection **(F)**.

(68%), Asia (17%), and Europe (11%). The patient's age ranged from 2 to 85 years (mean, 55.9; median, 56 yr.) with female average age slightly older than male. Of available cases, 80% is found incidentally without related symptoms (Figure 4C), and the remaining adopts pain, palpable mass, paresthesia, pleural effusion, neurologic deficits, hematuria, and other concurrent tumors (hepatic, renal, ovarian, endometrial carcinoma, etc.). Among all renal AHs, 24.4% associated with end-stage renal disease (ESRD). [4,18,25,30,31,34,35] Various involved anatomic locations were has been appreciated with the majority developing in kidney (n=51, 38.3%), spinal/paraspinal region (n=20, 15%), adrenal gland (n=18, 13.5%), and liver (n=9, 6.8%) (**Figure 4D**).

AH is usually solitary, but multiple lesions are also appreciable (Figure 4E); locations of the latter include kidney (45%), liver (20%), adrenal gland (10%), intestine (10%), paraspinal region (5%), ovary (5%), and breast (5%). The tumor can be solid, cystic, and solid-cystic in either imaging or gross examination (**Figure 4F**), ranging in size from 0.5 to 12 cm (mean, 3.0 cm; median, 2.6 cm; **Figure 4G**). Identical to the most reported cases, all tumors in our series were asymptomatic and found incidentally. As depicted in Figure 4H, the reported ways of resection are listed as follows by decreasing percentage: simple lesion resection (47%), organ resection (26%), partial organ resection (18%), radical resection (9%). Almost all the patients were alive without evidence of disease during the follow-up (range: 1 to 156 months; median, 14 months; mean,

26.2 months), except that one patient was dead after 107 months since AH arising in the explant was found.

## DISCUSSION

Hemangioma is a common soft tissue lesion and frequently arise in skin and subcutis.[7] The renal or adrenal hemangiomas are relatively rare, some of which have underlying other vascular diseases, such as Sturge-Weber syndrome, Klippel-Trenaunay syndrome, von Hippel-Lindau syndrome or systemic angiomatosis. [36-39] AH is an extremely rare benign tumor. Although cases of AHs have been continuously reported since 2009, most arein the form of case reports, many clinicians were still unfamiliar with this entity. Originally, it is uncertain whether AHs represent a bona fide endothelial tumor or reactive or malformation.[7] However, from the views of the pathologist, the distinct morphologies resemble hemangioma, suggesting their tumor nature[7]. Recently, recurrent GNAQ and GNA14 mutations were discovered in AH, supporting a role for this pathway in the pathogenesis of AHs and further indicating its true tumor nature.[11,28]

Clinically, unlike the other vascular lesions, AHs tend to be asymptomatic without typical manifestation such as hematuria or flank pain[40]. Therefore, it's difficult to early find this lesion unless in few patients associated with some symptoms or ESRD who need additional renal examination. Radiologically, non-contrast CT may show hypodense solid or cystic lesions and contrast-enhanced CT usually exhibits homogeneous enhancement or heterogeneous enhancement in the periphery. [9,16,24,33] However, imaging also seems not to help in differentiating AHs from other mimickers[15], although the angiosarcomas or renal cell carcinomas commonly demonstrate central necrosis that large AHs still can have.[32] The rarely possible pre-operative diagnosis by radiologic studies may give rise to clinically conservative management.[16] Laboratory biologic markers do not also help to recognize this disease. Nonetheless, the risk of AHs in patients who suffer from the chronic renal disease should be noted, due to its association with ESRD, particularly when there are relevant symptoms. Histologically, AH is typically well-demarcated without evidence of peripherally infiltration and neoplastic necrosis, the features practically pathognomonic when making a diagnosis.[4] It is characterized by interconnected sinusoidal capillary-like vessels lined by hobnail or flat endothelial cells with no obvious atypia, multilayering, and mitosis, although focal regions may demonstrate dilated cavernous-like vasculature.[9,30,35] Some cases have prominent sclerotic areas and hence form an alternating acellular and hypocellular pattern. [33] Mast cells with dark granules sometimes could be appreciable, which is likely to be considered as atypical endothelium resulting in a misdiagnosis of malignancies.[7,34]) In the peripheral regions of AHs, one usually finds the thrombotic fibrin-filled vessels, another feature of this entity.[7] Tumor cells prototypically stain positive with endothelial markers such as CD31, CD34, ERG, factor VIII-related protein.

The flagship diagnostic differentiation from AH is well-differentiated angiosarcoma due to its deficiency in limited atypia at times. The prominent anastomosing growth pattern is traditionally considered as a practically pathognomonic feature of angiosarcoma. The Morphologic overlap between these two may facilitate diagnostic pitfalls, particularly for the junior pathologist or in the small biopsy. In a contrast to well-differentiated angiosarcoma, AH does not show prominent nuclear atypia, increased proliferative activity, and mitosis, as well as multilayering of tumor cells.[7,13] Necrosis, other than some cases with regressive changes, is usually not present in AH.[13] However, a subset of highly differentiated sarcomas only shows very little atypia thoroughly, which may obscure their true malignant nature and give rise to erroneous diagnosis when telling AHs apart from angiosarcomas. The most crucial and consistent features are that contrary to AHs, angiosarcomas always have an infiltrative border with extensive destruction of renal parenchymal, peripheral collagen or adipose tissue.[4] Papillary endothelial hyperplasia (Masson's tumor) can also form loosely anastomotic vascular structures mimicking AH or angiosarcoma, but the intravascular growth and reactive vessel wall can provide useful diagnostic clues[41].

Concerning the treatment to AHs, the mainstay in the surgical resection. As discussed above, the primary surgical way of AHs is lesion resection only due to the difficulty to recognize their true nature of aggressiveness. No matter what surgical procedures were taken, almost all patients lead a calm period in the follow-up. Anyway, the long-term follow-up data further support the bona fide benign biologic behavior of AH that warrants the surgeon's consideration to avoid unnecessary overtreatments, such as radical resection, extended lymph node dissection, and unnecessary pre-/post-operative chemotherapy or radiotherapy[42,43]. However, there has been no consensus on the treatment for AHs. If the biopsy diagnosis can be established, the concrete treatments such as embolization, lesion resection, partial or total nephrectomy may depend on the lesion size, location, and presence of symptoms[32]. In our series, the surgical ways included lesion resection, organ resection, partial organ resection, none of the patients have evidence of recurrence, metastasis, and death, further indicating the innocent nature of AH.

## CONCLUSIONS

Renal or adrenal AH is a very rare vascular tumor. They have distinctive histologic features and favorable prognosis but mimic well-differentiated angiosarcoma. To be familiar with this entity contributes to avoiding diagnostic pitfalls and dilemmas and hence unnecessary overtreatment.

## CONFLICT OF INTEREST

The authors have disclosed that they have no significant relationship with or financial interest in any commercial companies as to this article.

## FUNDING

Supported by the Shanghai Science and Technology commission (17ZR1417500).

## REFERENCES

1. Habeeb O, Rubin BP. The Molecular Diagnostics of Vascular Neoplasms. Surg Pathol Clin. 2019;12:35-49.
2. Sepulveda A, Buchanan EP. Vascular tumors. Semin Plast Surg. 2014;28:49-57.
3. Antonescu C. Malignant vascular tumors--an

update. Mod Pathol. 2014;27 Suppl 1:S30-8.
4. Brown JG, Folpe AL, Rao P, et al. Primary vascular tumors and tumor-like lesions of the kidney: a clinicopathologic analysis of 25 cases. Am J Surg Pathol. 2010;34:942-9.
5. Zheng W, Fung KM, Cheng L, Osunkoya AO. Benign vascular tumors, cysts, and pseudocysts of the adrenal gland: a contemporary multi-institutional clinicopathological analysis of 55 cases. Hum Pathol. 2018;82:95-102.
6. Montgomery E, Epstein JI. Anastomosing hemangioma of the genitourinary tract: a lesion mimicking angiosarcoma. Am J Surg Pathol. 2009;33:1364-9.
7. John I, Folpe AL. Anastomosing Hemangiomas Arising in Unusual Locations: A Clinicopathologic Study of 17 Soft Tissue Cases Showing a Predilection for the Paraspinal Region. Am J Surg Pathol. 2016;40:1084-9.
8. Lin J, Bigge J, Ulbright TM, Montgomery E. Anastomosing hemangioma of the liver and gastrointestinal tract: an unusual variant histologically mimicking angiosarcoma. Am J Surg Pathol. 2013;37:1761-5.
9. Jin LU, Liu J, Li Y, et al. Anastomosing hemangioma: The first case report in the bladder. Mol Clin Oncol. 2016;4:310-2.
10. Tran TAN, Linos K, Carlson JA, Bridge JA. A primary cutaneous vascular neoplasm with histologic features of anastomosing hemangioma. J Cutan Pathol. 2019.
11. Bean GR, Joseph NM, Gill RM, Folpe AL, Horvai AE, Umetsu SE. Recurrent GNAQ mutations in anastomosing hemangiomas. Mod Pathol. 2017;30:722-7.
12. Peng X, Li J, Liang Z. Anastomosing haemangioma of liver: A case report. Mol Clin Oncol. 2017;7:507-9.
13. Dundr P, Nemejcova K, Laco J, et al. Anastomosing Hemangioma of the Ovary: A Clinicopathological Study of Six Cases with Stromal Luteinization. Pathol Oncol Res. 2017;23:717-22.
14. Kishida N, Sentani K, Terada H, et al. Anastomosing haemangioma with fatty changes in the perirenal space: a lesion mimicking liposarcoma. BJR Case Rep. 2018;4:20170022.
15. O'Neill AC, Craig JW, Silverman SG, Alencar RO. Anastomosing hemangiomas: locations of occurrence, imaging features, and diagnosis with percutaneous biopsy. Abdom Radiol (NY). 2016;41:1325-32.
16. Al-Maghrabi HA, Al Rashed AS. Challenging Pitfalls and Mimickers in Diagnosing Anastomosing Capillary Hemangioma of the Kidney: Case Report and Literature Review. Am J Case Rep. 2017;18:255-62.
17. Omiyale AO, Golash A, Mann A, Kyriakidis D, Kalyanasundaram K. Anastomosing Haemangioma of the Kidney Involving a Segmental Branch of the Renal Vein. Case Rep Surg. 2015;2015:927286.
18. Abboudi H, Tschobotko B, Carr C, DasGupta R. Bilateral Renal Anastomosing Hemangiomas: A Tale of Two Kidneys. J Endourol Case Rep. 2017;3:176-8.
19. Tahir M, Folwell A. Anastomosing haemangioma of kidney: a rare subtype of vascular tumour of the kidney mimicking angiosarcoma. ANZ J Surg. 2016;86:838-9.
20. Gunduz M, Hurdogan O, Onder S, Yavuz E. Cystic Anastomosing Hemangioma of the Ovary: A Case Report With Immunohistochemical and Ultrastructural Analysis. Int J Surg Pathol. 20181066896918817148.
21. Zhao M, Li C, Zheng J, Sun K. Anastomosing hemangioma of the kidney: a case report of a rare subtype of hemangioma mimicking angiosarcoma and review of the literature. Int J Clin Exp Pathol. 2013;6:757-65.
22. Brehm B, Rauh C, Dankerl P, Schulz-Wendtland R. [Anastomosing hemangioma in the male breast -- a rarity]. Rofo. 2014;186:80-1.
23. Caballes AB, Abelardo AD, Farolan MJ, Veloso JAD. Pediatric Anastomosing Hemangioma: Case Report and Review of Renal Vascular Tumors in Children. Pediatr Dev Pathol. 20181093526618809230.
24. Heidegger I, Pichler R, Schafer G, et al. Long-term follow up of renal anastomosing hemangioma mimicking renal angiosarcoma. Int J Urol. 2014;21:836-8.
25. Ross M, Polcari A, Picken M, Sankary H, Milner J. Anastomosing hemangioma arising from the adrenal gland. Urology. 2012;80:e27-8.
26. Wetherell DR, Skene A, Manya K, Manecksha RP, Chan Y, Bolton DM. Anastomosing haemangioma of the kidney: a rare morphological variant of haemangioma characteristic of genitourinary tract location. Pathology. 2013;45:193-6.
27. Tran TA, Pernicone P. Anastomosing hemangioma with fatty changes of the genitourinary tract: a lesion mimicking angiomyolipoma. Cent European J Urol. 2012;65:40-2.
28. Bean GR, Joseph NM, Folpe AL, Horvai AE, Umetsu SE. Recurrent GNA14 mutations in anastomosing haemangiomas. Histopathology. 2018;73:354-7.
29. Zhao M, Kong M, Yu JJ, He XL, Zhang DH, Teng XD. [Clinicopathologic analysis of anastomosing hemangioma of the kidney and adrenal gland]. Zhonghua Bing Li Xue Za Zhi. 2016;45:698-702.
30. Kryvenko ON, Gupta NS, Meier FA, Lee MW, Epstein JI. Anastomosing hemangioma of the genitourinary system: eight cases in the kidney and ovary with immunohistochemical and ultrastructural analysis. Am J Clin Pathol. 2011;136:450-7.
31. Mehta V, Ananthanarayanan V, Antic T, et al. Primary benign vascular tumors and tumorlike lesions of the kidney: a clinicopathologic analysis of 15 cases. Virchows Arch. 2012;461:669-76.
32. Silva MA, Fonseca E, Yamauchi FI, Baroni

RH. Anastomosing hemangioma simulating renal cell carcinoma. Int Braz J Urol. 2017;43:987-9.

33. Tao LL, Dai Y, Yin W, Chen J. A case report of a renal anastomosing hemangioma and a literature review: an unusual variant histologically mimicking angiosarcoma. Diagn Pathol. 2014;9:159.
34. Perdiki M, Datseri G, Liapis G, et al. Anastomosing hemangioma: report of two renal cases and analysis of the literature. Diagn Pathol. 2017;12:14.
35. Chou S, Subramanian V, Lau HM, Achan A. Renal Anastomosing Hemangiomas With a Diverse Morphologic Spectrum: Report of Two Cases and Review of Literature. Int J Surg Pathol. 2014;22:369-73.
36. Schofield D, Zaatari GS, Gay BB. Klippel-Trenaunay and Sturge-Weber syndromes with renal hemangioma and double inferior vena cava. J Urol. 1986;136:442-5.
37. Yavas GF, Okur N, Kusbeci T, Norman E, Inan U. A case of von Hippel-Lindau disease with juxtapapillary retinal capillary hemangioma and nutcracker phenomenon. Int Ophthalmol. 2013;33:309-14.
38. Zemni I, Haddad S, Hlali A, Manai MH, Essoussi M. Adrenal gland hemangioma: A rare case of the incidentaloma: Case report. Int J Surg Case Rep. 2017;41:417-22.
39. Seppala A, Olow B. Renal angiomatosis with fatal perirenal haemorrhage. Case report. Acta Chir Scand. 1972;138:636-8.
40. Soleimani MJ, Shadpour P, Mehravaran K, Kashi AH. Laser Treatment for Urethral Hemangiomas: Report of Three Cases. Urol J. 2017;14:3094-9.
41. Hashimoto H, Daimaru Y, Enjoji M. Intravascular papillary endothelial hyperplasia. A clinicopathologic study of 91 cases. Am J Dermatopathol. 1983;5:539-46.
42. Capitanio U, Becker F, Blute ML, et al. Lymph node dissection in renal cell carcinoma. Eur Urol. 2011;60:1212-20.
43. Bindayi A, Hamilton ZA, McDonald ML, et al. Neoadjuvant therapy for localized and locally advanced renal cell carcinoma. Urol Oncol. 2018;36:31-7.